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A MANUAL

of the

PRACTICE of MEDICINE

PREPARED ESPECIALLY FOR STUDENTS

BY

A. A. STEVENS, A. M., M. D.

Professor of Therapeutics and Clinical Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Medicine in the University of Pennsylvania; Physician to the Philadelphia General Hospital, to the Episcopal Hospital, and to St. Agnes' Hospital; Fellow of the College of Physicians of Philadelphia, etc.

"——is an arch wherethro'
Gleams that untravell'd world whose margin fades
Forever and forever when I move."

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ILLUSTRATED

PHILADELPHIA AND LONDON
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PREFACE TO THE TENTH EDITION

In preparing the present edition of this book the text has been carefully revised. Some of the chapters have been entirely rewritten; a number of new ones have been added; there is scarcely one that has not been enlarged or otherwise changed. The more important modifications will be found in the sections relating to leukemia, chronic nephritis, pyelonephritis, cardiac arhythmia, pulmonary tuberculosis, typhus fever, malaria, pyogenic infections, bacillary dysentery, diabetes mellitus, tetany, locomotor ataxia, heat stroke, and dermatitis exfoliativa. Also articles relating to Vincent's angina, diverticula of the esophagus, acholuric jaundice, embolism and thrombosis of the mesenteric vessels, alkaptonuria, albumosuria, tests of the functional capacity of the kidneys, polycystic disease and malignant disease of the kidney, chronic enlargements of the spleen, hypopituitarism, infantilism, glandular fever, Rocky Mountain spotted fever, Weil's disease, Kala-azar, congenital amyotonia, and myasthenia gravis, have been added. The plan of the book remains unchanged.

June, 1915. A. A. S.

41876

CONTENTS

DISEASES OF THE DIGESTIVE SYSTEM

PAGE
General Symptomatology—
The Teeth and Gums 17
The Tongue 17
Discoloration of the Tongue 18
Enlargement of the Tongue 18
Tremor of the Tongue 19
Fissures on the Tongue 19
Scars on the Tongue 19
Fetor of the Breath 19
The Appetite 19
Dysphagia 20
Vomiting, or Emesis 20
The Vomit 20
Examination of the Gastric Contents 21
Acidity of the Gastric Contents 25
Rumination, or Merycismus 25
Hiccup 26
The Stools 26
Abdominal Distention 27
Diseases of the Mouth, Tonsils, Pharynx, and Esophagus—
Stomatitis 28
Acute Catarrhal Stomatitis 28
Aphthous Stomatitis 29
Ulcerative Stomatitis 29
Mycotic Stomatitis 29
Gangrenous Stomatitis 30
Mercurial Stomatitis 31
Gonorrheal Stomatitis 31
Acute Tonsillitis 31
Hypertrophy of the Tonsils 34
Pharyngitis 35
Acute Pharyngitis 35
Vincent's Angina 36
Ludwig's Angina 37
Chronic Pharyngitis 37
Retropharyngeal Abscess 38
Stenosis of the Esophagus 39
Spasm of the Esophagus 39
Organic Esophageal Obstruction 39
Diverticula of the Esophagus 40

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General Symptomatology (*Continued*)—

PAGE
Plethora 163
Hydremia 163
Anhydremia 163
Melanemia 163
Polycythemia 163
Microcytosis and Macrocytosis 164
Poikilocytosis 164
Nucleated Red Cells 164
Leukocytosis 164
Eosinophilia 165
Leukopenia, or Hypoleukocytosis 165
Lipemia 165
Blood Parasites 165
Oligochromemia 166
Oligocythemia 166
Anemia 166
Secondary Anemia 167
Primary Anemia 167
Pernicious Anemia 168
Chlorosis 170
Leukemia 171
Hodgkin's Disease 174
Hemophilia 175
Purpura 176
Secondary Purpura 176
Primary Purpura 177
Erythremia 178

Diseases of the Ductless Glands—

Addison's Disease 180
Exophthalmic Goiter 181
Myxedema; Hypothyroidism 183
Acromegaly 185
Hypopituitarism 186
Tetany 187
Primitive Splenomegaly with Anemia 189
Other Chronic Enlargements of the Spleen 189

DISEASES OF THE CIRCULATORY SYSTEM

General Symptomatology—

The Apex-beat 191
Displacement of the Apex-beat 192
Changes in the Force and Extent of the Apex-beat 192
Abnormal Centers of Pulsation 192
Precordial Prominence 193
Palpation 194
Percussion 194
Auscultation 195
The Intensity of the Heart-sounds 195
Alteration in the Rhythm of the Heart-sounds 196
Adventitious Sounds, or Murmurs 196
Pericardial Friction-sound 196
uropericardial Friction-sound 197

General Symptomatology (*Continued*)—

PAGE
Local Prominences and Depressions 241
Expansion 242
Litten's Diaphragm Phenomenon 242
Palpation 243
Percussion 244
Auscultation 246
Mensuration 250
Radioscopy 250

Diseases of the Nose and Larynx—

Acute Coryza 251
Chronic Nasal Catarrh 252
Acute Catarrhal Laryngitis 254
Spasmodic Group 256
Membranous Croup 257
Chronic Laryngitis 257
Laryngismus Stridulus 259
Edema of the Larynx 260

Diseases of the Bronchi and Lungs—

Bronchitis 262
Acute Catarrhal Bronchitis 262
Chronic Bronchitis 265
Fibrinous Bronchitis 267
Bronchiectasis 268
Asthma 270
Essential Asthma 270
Hay Asthma 274
Pulmonary Emphysema 275
Hypertrophic Emphysema 275
Hemoptysis 278
Hemorrhagic Infarct of the Lung 279
Congestion of the Lungs 280
Hypostatic Congestion of the Lungs 281
Edema of the Lungs 282
Croupous Pneumonia 283
Bronchopneumonia 289
Chronic Interstitial Pneumonia 294
Abscess of the Lung 295
Gangrene of the Lung 296
Pulmonary Tuberculosis 297

Diseases of the Pleura—

Pleurisy 308
Empyema 312
Hydrothorax 313
Pneumothorax 314
Hemothorax 315

ACUTE INFECTIOUS DISEASES

Fever 316
Period of Incubation 319
Date at which Rashes Appear 319
Protection from Future Attacks 320
Protracted Cases of Fever 320

Acute Infectious Diseases (*Continued*)—

PAGE
Infections in which Jaundice is Likely to Occur . . . 321
Subnormal Temperature . . . 321
Febricula . . . 321
Typhoid Fever . . . 322
Typhus Fever . . . 332
Relapsing Fever . . . 334
Cerebrospinal Fever . . . 336
Malarial Fever . . . 339
Intermittent Malarial Fever . . . 342
Estivo-autumnal Fever . . . 343
Pernicious Malarial Fever . . . 343
Malarial Cachexia . . . 344
Scarlet Fever . . . 347
Measles . . . 352
Rubella . . . 354
Smallpox . . . 355
Vaccinia . . . 360
Varicella . . . 361
Diphtheria . . . 362
Rheumatic Fever . . . 367
Erysipelas . . . 371
Pyogenic Infections . . . 374
Influenza . . . 377
Mumps . . . 379
Yellow Fever . . . 380
Acute General Tuberculosis . . . 383
Whooping-cough . . . 384
Cholera . . . 387
Dysentery . . . 391
Bacillary Dysentery . . . 391
Amebic Dysentery . . . 394
Tetanus . . . 395
Epidemic Poliomyelitis . . . 397
Dengue . . . 400
Hydrophobia . . . 401
Leprosy . . . 402
Plague . . . 404
Glandular Fever . . . 406
Rocky Mountain Spotted Fever . . . 406
Kala-azar . . . 407
Malta Fever . . . 407
Sleeping Sickness . . . 408

WORM-INFECTION—HELMINTHIASIS

Tapeworm or Cestode Infection . . . 409
Round-worm or Nematode Infection . . . 411

DISORDERS OF METABOLISM

Gout . . . 415
Rickets . . . 419
Diabetes Mellitus . . . 421

Disorders of Metabolism (*Continued*)— PAGE

Diabetes Insipidus 425
Scurvy 427
Beriberi 428

DISEASES OF THE NERVOUS SYSTEM

Disturbances of Motion

Paralysis 430
Irregular Paralysis 430
Monoplegia 431
Hemiplegia 431
Paraplegia 432
Convulsions 433
General Clonic Convulsions 434
Tonic Convulsions 434
Hysteric Convulsions 434
Eclampsia 435
Local Convulsions 435
Choreiform Movements 435
Athetosis 437
Tremors 437
The Gait 437
The Reflexes 438

Disturbances of Sensation

Anesthesia 440
Hemianesthesia 440
Monanesthesia 440
Paranesthesia 441
Analgesia and Thermo-anesthesia 441
Astereognosis 441
The Sense of Space 441
Hyperesthesia 441
Paresthesia 441
Neuralgia 442
Muscle Sense 442

Disturbances of Nutrition

Muscular Atrophy 442
Changes in Electromuscular Contractility 442
Arthropathies 443
Ulceration Resulting from Perverted Nutrition 443

Disturbances of Consciousness

Coma 444
Catalepsy 445

Disturbances of the Special Senses

The Eye 446
The Ear 447

Psychic Disturbances

PAGE
Delusion . . . 447
Illusion . . . 448
Hallucination . . . 448
Imperative Conceptions . . . 448
Morbid Impulse . . . 448
Delirium . . . 448

DISEASES OF THE BRAIN, SPINAL CORD, AND NERVES

Acute Cerebral Leptomeningitis . . . 450
Chronic Cerebral Leptomeningitis . . . 452
Chronic Cerebral Pachymeningitis . . . 453
Hemorrhagic Pachymeningitis . . . 453
Chronic Hydrocephalus . . . 454
Paretic Dementia . . . 455
Cerebral Paralysis in Children . . . 458
Cerebral Hyperemia . . . 459
Cerebral Anemia . . . 460
Cerebral Hemorrhage . . . 461
Obstruction of the Cerebral Arteries . . . 466
Morbid Growths in the Brain . . . 467
Abscess of the Brain . . . 472
Aphasia . . . 473
Spinal Leptomeningitis . . . 474
Hypertrophic Cervical Pachymeningitis . . . 476
Myelitis . . . 476
Chronic Anterior Poliomyelitis . . . 480
Primary Spastic Paraplegia . . . 481
Amyotrophic Lateral Sclerosis . . . 482
Bulbar Paralysis . . . 483
Acute Ascending Paralysis . . . 484
Locomotor Ataxia . . . 485
Ataxic Paraplegia . . . 489
Disseminated Cerebrospinal Sclerosis . . . 489
Hereditary Ataxia . . . 490
Syringomyelia . . . 491
Caisson Disease . . . 492
Localized Neuritis . . . 493
Multiple Neuritis . . . 494
Sciatica . . . 496
Facial Paralysis . . . 497

Functional Nervous Diseases—

Headache . . . 499
Vertigo . . . 503
Ménière's Disease . . . 504
Epilepsy . . . 504
Hysteria . . . 508
Neurasthenia . . . 512
Acute Chorea . . . 514
Neuralgia . . . 516
Migraine . . . 520
Paralysis Agitans . . . 521
Occupation Neuroses . . . 523

DISEASES OF THE MUSCLES AND JOINTS

PAGE
Myalgia . . . 524
Muscular Dystrophies . . . 526
Congenital Myotonia . . . 527
Congenital Amyotonia . . . 528
Myasthenia Gravis . . . 528
Arthritis Deformans . . . 528
Chronic Rheumatism . . . 531

VASOMOTOR AND TROPHIC DISORDERS, SUNSTROKE, AND INTOXICATIONS

Raynaud's Disease . . . 532
Angioneurotic Edema . . . 533
Heat Stroke . . . 533
Alcoholism . . . 534
Chronic Opium Poisoning . . . 537
Chronic Lead-poisoning . . . 537
Pellagra . . . 539

DISEASES OF THE SKIN AND ITS APPENDAGES

General Symptomatology—

The Color of the Skin . . . 540
Hardness or Induration of the Skin . . . 540
Glossy Skin . . . 541
Enlargement of the Superficial Veins . . . 541
Cutaneous Emphysema . . . 541
Abnormal Conditions of the Nails . . . 541
Atrophy of the Nails . . . 541
Curving of the Nails . . . 542
Onychia . . . 542

Cutaneous Eruptions—

Macules . . . 542
Diffuse Erythema . . . 544
Vesicles . . . 544
Blebs or Bullæ . . . 546
Pustules . . . 546
Papules . . . 548
Tubercles . . . 549
Wheals or Pomphi . . . 550
Crusts . . . 550
Scales . . . 551
Ulcers . . . 552

Diseases of the Sweat-glands—

Hyperidrosis . . . 554
Bromidrosis . . . 555
Chromidrosis . . . 555
Sudamen . . . 555

Functional Diseases of the Sebaceous Glands—

Seborrhea . . . 556
Comedo . . . 558
Milium . . . 559
Steatoma . . . 559

Inflammatory Diseases of the Skin— PAGE

Erythema Simplex . . . 560
Erythema Intertrigo . . . 560
Erythema Nodosum . . . 561
Erythema Multiforme . . . 561
Urticaria . . . 562
Herpes Simplex . . . 564
Herpes Zoster . . . 565
Acne Vulgaris . . . 565
Acne Rosacea . . . 568
Sycosis . . . 568
Furunculus . . . 570
Carbunculus . . . 570
Psoriasis . . . 571
Eczema . . . 573
Lichen Ruber, Lichen Planus, and Lichen Scrofulosis . . . 576
Prurigo . . . 577
Dermatitis Herpetiformis . . . 577
Dermatitis . . . 578
Ecthyma . . . 581
Pemphigus . . . 582
Impetigo Contagiosa . . . 583
Miliaria . . . 584
Pompholyx . . . 585

Atrophic Affections of the Skin—

Albinism . . . 585
Vitiligo . . . 586

Atrophic Affections of the Hair and Nails—

Alopecia . . . 586
Alopecia Areata . . . 587

Hypertrophic Affections of the Skin—

Lentigo . . . 588
Chloasma . . . 589
Keratosis Pilaris . . . 589
Molluscum Epitheliale . . . 590
Callositas . . . 591
Clavus . . . 591
Cornu Cutaneum . . . 592
Verruca . . . 592
Nævus Pigmentosus . . . 593
Ichthyosis . . . 594

Hypertrophic Affections of the Hair and Nails—

Hypertrichosis . . . 594
Scleroderma . . . 594
Morphea . . . 595
Elephantiasis . . . 595

New Growths of the Skin—

Keloid . . . 596
Fibroma . . . 597
Angioma . . . 598
Xanthoma . . . 598
Lupus Erythematosus . . . 599
Lupus Vulgaris . . . 600
Syphilis Cutanea . . . 601

New Growths of the Skin (*Continued*)— PAGE
Epithelioma 604
Ainhum 606
Neuroses of the Skin—
Pruritus 606
Parasitic Affections of the Skin—
Ringworm 607
Tinea Tonsurans 608
Tinea Circinata 609
Tinea Sycosis 609
Tinea Versicolor 610
Favus 611
Scabies 611
Pediculosis 612

INDEX 615

A MANUAL

OF THE

PRACTICE OF MEDICINE

DISEASES

OF THE

DIGESTIVE SYSTEM

THE TEETH AND GUMS

Delayed dentition and the eruption of badly formed teeth may result from rickets, congenital syphilis, or cretinism.

Hutchinson's Teeth.—The lateral incisors of the upper jaw are pegged, and the central incisors of the same jaw have convex sides and crescentic notches on their cutting-edges. These peculiarities indicate hereditary syphilis, and are noted only in the permanent teeth.

A blue line on the gums near the insertion of the teeth usually indicates chronic lead-poisoning. Copper- and silver-poisoning occasionally produce similar lines.

Spongy, bleeding gums are often associated with scurvy. Swelling of the gums, with tenderness and salivation, occurs in mercurial poisoning.

THE TONGUE

Fur on the Tongue.—This consists for the most part of accumulated epithelial cells, particles of food, and micro-organisms, and occurs in a great variety of diseases, both local and general.

A light, uniform coat is often noted in health, particularly in those who sleep with the mouth open. Other casual conditions are: (1) Febrile diseases. (2) Dyspepsia. (3) Catarrhal conditions of the nose and throat. Very little diagnostic significance is to be attached to the appearance of the tongue in diseases of the stomach.

Circumscribed furring often indicates local disturbance, as a jagged tooth or tonsillitis.

Unilateral furring may result from disturbed innervation, as in conditions affecting the second and third branches of the fifth nerve. It has been noted in neuralgia of those branches, and in fractures of the skull involving the foramen rotundum.

The dry, brown and *fissured tongue* is noted in asthenic fevers, as typhoid fever, pneumonia, and septicopyemia.

A red, beefy tongue is sometimes noted in chronic wasting diseases. It is of quite frequent occurrence in dysentery and in diabetes.

The "strawberry tongue" is characterized by a white fur, through which project bright red and prominent papillæ. It is seen in the early stage of scarlet fever.

DISCOLORATION OF THE TONGUE

Black Tongue (*Nigrities Linguæ*).—This is a parasitic affection of the tongue, characterized by the appearance of black patches on the center of the dorsum, with great prolongation of the filiform papillæ.

Bluish-black discoloration of the tongue is observed in Addison's disease.

Leukoplakia Buccalis.—In this condition there are slightly elevated, smooth, opaque, whitish plaques on the lingual or buccal mucous membrane. There are no subjective symptoms. Excessive smoking is a common cause. Syphilis appears to be a factor in some cases. It is sometimes associated with chronic affections of the skin, notably psoriasis. Epithelioma of the mouth is not an uncommon sequel.

ENLARGEMENT OF THE TONGUE

Macroglossia, or chronic enlargement of the tongue, occurs in myxedema (cretinism), acromegaly, and occasionally in syphilis.

TREMOR OF THE TONGUE

Trembling of the tongue is noted in many conditions; it is particularly marked in asthenic fevers (typhoid), in alcoholism, and in paretic dementia.

FISSURES ON THE TONGUE

Fissures on the tongue may result from severe glossitis, syphilis, carcinoma, tuberculosis, or the impact of a jagged tooth.

SCARS ON THE TONGUE

Scars on the tongue often result from syphilitic lesions or from the tooth wounds of epilepsy.

FETOR OF THE BREATH

This is often due to local inflammation, as chronic rhinitis, pyorrhea alveolaris, tonsillitis, etc., to the retention of decomposing food, to caries of the teeth, to certain lung diseases, especially gangrene and bronchiectasis, to dyspepsia, and to the ingestion of certain foods or drugs.

THE APPETITE

Bulimia, or *inordinate appetite*, is a common symptom in nervous dyspepsia, hysteria, diabetes, and in certain insanities, notably in paretic dementia. It may be due to intestinal parasites.

Anorexia, or *loss of appetite*, is a symptom common to many conditions.

Parorexia is a craving for special articles of diet. *Pica* is a desire for articles that are not foods. These perversions of the appetite are noted particularly in hysteria, insanity, chlorosis, pregnancy, and helminthiasis.

DYSPHAGIA

Dysphagia, or difficult swallowing, may result from: (1) Local inflammation, especially tuberculous ulceration of the throat or larynx. (2) Stricture due to the healing of an ulcer (corrosive poisons, syphilis, typhoid fever). (3) Cancer of the esophagus. (4) Spasm of the esophagus (hydrophobia, hypochondriasis, hysteria, etc.). (5) A foreign body. (6) Pressure on the esophagus (aneurysm, mediastinal tumor, enlarged glands, pericardial effusion). (7) Paralysis, local, as in diphtheritic neuritis, or centric, as in bulbar disease.

VOMITING, OR EMESIS

Etiology.—(1) Toxic, from ptomains, drugs, uremia, and the specific fevers. (2) Centric disease, as cerebral tumors and meningitis; this type is often unaccompanied with nausea, and does not relieve the associated headache. (3) Diseases of the stomach, as ulcer, cancer, dilatation, catarrh, etc. (4) Reflex, as from pregnancy, uterine or ovarian disease, irritation of the fauces, worms, biliary colic, etc. (5) Intestinal obstruction (often fecal). (6) Disturbed cerebral circulation, as in swinging and in sea-sickness. (7) Incessant coughing, as in whooping-cough or advanced pulmonary tuberculosis. (8) Certain neuroses, as hysteria, migraine. (9) Periodic vomiting may be in itself a neurosis, may form a part of the gastric crises of locomotor ataxia or may occur in acidosis (recurrent vomiting of infants). (10) Esophageal vomiting results from obstruction, and the vomit is alkaline in reaction.

THE VOMIT

Watery or mucous vomit is noted in chronic gastritis, in certain forms of nervous dyspepsia, in cerebral disease, and after persistent emesis, as in cholera.

Bilious or green vomit is not diagnostic of any special condition; it may occur in any case in which there are persistent vomiting and retching.

Bloody Vomit (*Hematemesis*).—For causes, see page 66. If present in large amount, the blood can usually be recognized by the unaided eye; small amounts may be detected by the microscope, by the spectroscope, or by chemical tests.

Purulent vomit may result from the rupture of an abscess into the esophagus or stomach or from phlegmonous gastritis.

Fecal vomit (*stercoraceous vomit*) indicates intestinal obstruction or a gastro-colic fistula, the result of ulcer or cancer. It is recognized by its odor and appearance.

Profuse Vomit.—The ejection of enormous quantities of frothy fermented material at intervals is highly significant of gastric dilatation.

Vomiting without nausea, distress, or other gastric phenomena occurs in certain neuroses of the stomach, in hysteria, uremia, and in brain disease, as tumor or as a precursor of apoplexy.

EXAMINATION OF THE GASTRIC CONTENTS

The *test-breakfast* of Ewald and Boas consists of a roll and from 10 to 14 fluidounces of water or weak tea. It is given in the morning on an empty stomach, and is removed in one hour by aspiration or expression. The roll should be thoroughly masticated. This breakfast affords a satisfactory means of determining the secretory activity of the stomach. Riegel's *test-meal*, however, is better adapted to determining the total functional activity of the stomach. It consists of a plate of meat-broth, a beef-steak weighing from 5 to 7 ounces, 1½ ounces of mashed potatoes, and a roll. The contents are removed in three or four hours after the ingestion of the meal.

Test for Free Acids.—Filter-paper soaked in a solution of Congo-red and dried turns blue in the presence of free acids. A saturated alcoholic solution of tropeolin 00 turns from a brownish yellow to a dark brown when brought in contact with fluids containing free acids.

Qualitative Tests for HCl.—Günzburg's phloroglucin-vanillin test will react with 1 part of HCl in 15,000 parts of water. The solution consists of 2 parts of phloroglucin, 1 part of vanillin, and 30 parts of absolute alcohol. When a few drops of this solution are heated with an equal quantity of the filtrate contained in a porcelain dish, a beautiful red color appears at the margin of the fluid. Boas states that the test is still more delicate when 100 parts of 80 per cent. alcohol are substituted for the 30 parts of absolute alcohol.

Boas' resorcin-sugar test gives a similar reaction. The reagent consists of 5 parts of resorcin, 3 parts of sugar, and 100 parts of diluted alcohol.

Total Acidity.—This is determined by allowing a decinormal alkali solution (water, 10 c.c.; potassium hydrate, 56 mg.) to flow from a buret, drop by drop, into a beaker containing 10 c.c. of filtered gastric juice, to which have been added as an indicator two drops of a 1 per cent. alcoholic solution of phenolphthalein. The test is completed when the red color produced no longer disappears on shaking the solution. Ten c.c. of normal gastric juice usually require from 4 to 6.5 c.c. of the standard alkali solution.

Since 1 c.c. of the alkali solution is equivalent to 0.00364 gram of HCl, it follows that the percentage of the latter in a given specimen will equal the number of cubic centimeters of the alkali solution required multiplied by 10, and again by 0.00364.

Quantitative Test for Free Hydrochloric Acid.—*Mintz's Color Method.*—To 10 c.c. of the filtrate add a decinormal solution of sodium hydrate from a buret until a droplet (removed with a platinum loop) of the fluid no longer reacts with Günzburg's reagent. The number of cubic centimeters of the alkaline solution used, multiplied by 10 and then by 0.00364, gives the percentage of free hydrochloric acid. This method, which is sufficiently accurate for clinical purposes, is based upon the supposition that the alkali first unites with the free acid before it affects the acid in organic combinations.

Test for Lactic Acid.—The presence of lactic acid in the stomach-contents simply indicates the existence of subacidity and of stagnation. These two conditions are never so constantly present nor so intense as in carcinoma (Riegel). When free HCl is present in sufficient quantities, it is unnecessary to test for lactic acid.

Uffelmann recommends a mixture of 10 c.c. of a 4 per cent. carbolic acid solution and 20 c.c. of distilled water, to which is added one drop of the official liquor ferri chloridi. This makes a clear amethyst-blue solution. The reagent must always be prepared at the time of making the test. If the solution turns yellowish green on the addition of filtered gastric contents, the presence of lactic acid is demonstrated. As the blue serves only as a contrast color, a very dilute solution of iron chlorid alone (one drop of liquor ferri chloridi in 50 c.c. of distilled water) suffices. As other substances, such as sugar, alcohol, acid phosphates, etc., give a somewhat similar reaction, the test is made more reliable by exhausting the gastric filtrate with pure ether (10 vol.), evaporating the ether, and adding the reagent to an aqueous solution of the residue.

Test for Acetic Acid.—This acid may be detected by its odor. The production of a blood-red color on the addition of a neutral solution of ferric chlorid to an aqueous solution of the ethereal extract, which has been neutralized with sodium carbonate, also indicates the presence of acetic acid.

Test for Butyric Acid.—This acid strikes a brownish-yellow color with Uffelmann's reagent. Its odor is also characteristic.

Tests for Rennet and Rennet Zymogen.—*Rennet.*—Add 5 c.c. of filtrate, which has been exactly neutralized with a decinormal alkaline solution, to an equal quantity of neutral raw or boiled milk, and keep the mixture at a temperature of about 100° F. If a flocculent coagulate forms in from ten to thirty minutes, the presence of rennet is revealed.

Rennet Zymogen.—Render 5 c.c. of filtrate very slightly alkaline with a 1 per cent. solution of sodium bicarbonate, add about 2 c.c. of a 1 per cent. solution of calcium chlorid, then mix with an equal quantity of pure milk, and keep the mixture at a temperature of 100° F. If zymogen is present, coagulation occurs in the usual time.

Test for Pepsin.—If free hydrochloric acid is present, the presence of pepsin in sufficient quantities may be assumed. To determine the presence of pepsin, pour 10 c.c. of filtrate into a test-tube. If free HCl is absent, add a sufficient quantity of acid to cause the appearance of the Congo reaction. Drop discs (1.5 mm. thick and 10 mm. in diameter) of hard-boiled egg into the mixture, and put the test-tube into the thermostat at 100° F. If sufficient pepsin is present, the discs will be completely dissolved in from one-half to one hour.

Test for Carbohydrates.—When starch digestion is arrested too early, as in cases of excessive secretion of HCl, Lugol's solution gives a blue or purple coloration with the gastric contents. Complete absence of color reaction indicates very active starch digestion (subacidity).

The Absorptive Power of the Stomach.—This is usually determined by the time required for free iodin to appear in the saliva after the ingestion of potassium iodid. The saliva is received on filter-paper impregnated with starch; a drop or two of fuming nitric acid is then added, and the appearance of a blue color proclaims the presence of iodin. Normally the saliva should yield the reaction for iodin in from ten to fifteen minutes after the ingestion of a capsule containing 0.1 gram of potassium iodid. Care must be taken that none of the drug adheres to the outside of the capsule. This test cannot be regarded as being very reliable.

The Motor Power of the Stomach.—Ewald has suggested the use of salol, which escapes from the stomach into the intestine, where it is broken up into salicylic acid and phenol. Normally salicyluric acid appears in the urine in from forty to seventy-five minutes after the ingestion of 1 gram of salol. Filter-paper moistened with urine containing salicyluric acid assumes a violet color when treated with a 10 per cent. ferric chlorid solution.

Riegel's test is more reliable. If it is found that seven hours after a test-meal of broth, beef-steak, mashed potatoes, and a roll (see p. 21) much food is still left in the stomach, the motor power is reduced. No remains of the *test-breakfast* should be found after two hours.

ACIDITY OF THE GASTRIC CONTENTS

Normal acidity is due to hydrochloric acid, but other acids are frequently formed during the digestive process, such as lactic, butyric, and acetic acids. The quantity of hydrochloric acid in normal gastric juice varies from 0.14 to 0.2 per cent., more acid being secreted after a heavy meal than after a light one. The amount of acidity per 100 c.c. of gastric contents is usually indicated by the number of cubic centimeters of $\frac{1}{10}$ normal sodium hydrate solution that are required to bind the acid. The average figures for *total acidity* are 50 to 75, and for *free* hydrochloric acid, 20 to 40.

Hyperacidity (hyperchlorhydria) results from a variety of causes. Mental overexertion, and the persistent use of highly seasoned foods are general predisposing factors. It is frequently present in neurasthenia and in hysteria. It may attend the gastric crises of locomotor ataxia. It may result from the abuse of tobacco. It is present, as a rule, in ulcer of the stomach. It is a common symptom in chlorosis. It sometimes occurs in cholelithiasis and in nephrolithiasis.

Subacidity and Anacidity (Hypochylia Gastrica and Achylia Gastrica).—Decreased secretion of gastric juice is seen in chronic gastritis; in gastric cancer; in atrophy of the gastric tubules; in passive congestion of the stomach; often in febrile diseases; often in severe anemia; and in certain neuroses, as neurasthenia, hysteria, and some forms of nervous dyspepsia.

RUMINATION, OR MERYCISMUS

Rumination is a condition, rarely observed in man, in which the food is regurgitated from the stomach and subjected to a second mastication. It is the result of a neurosis, and is generally found in association with hysteria, epilepsy, neurasthenia, or idiocy. It is sometimes hereditary or acquired by imitation.

HICCUP

Hiccup, or singultus, results from a clonic spasm of the diaphragm, and is often noted as a temporary condition after too rapid eating or drinking. Persistent hiccup is sometimes present in extreme exhaustion following acute or chronic diseases. It may also result from irritation of the phrenic nerve, as from the pressure of a thoracic aneurysm. It may be reflex from stomachic, hepatic, intestinal, or peritoneal disease. It may be due to hysteria.

THE STOOLS

Blood in the Stools (*Enterorrhagia or Melena*).—The blood is nearly normal in appearance after profuse hemorrhages, or when it has been quickly discharged, as in piles and fissure. Retained blood imparts a black or tarry appearance to the stools.

Melena may result from: (1) Traumatism; (2) acute inflammation of the bowels, as in enteritis and dysentery; (3) passive congestion, as in chronic heart and liver disease; (4) vicarious menstruation (extremely rare); (5) blood dyscrasia, as in scurvy, purpura, infectious fevers, etc.; (6) rupture of an aneurysm; (7) ulcers in the intestines, as peptic, typhoid, dysenteric, tuberculous or malignant ulcers; (8) intussusception; (9) the passage of blood from the stomach in hematemesis; (10) hemorrhagic infarction of the bowel from embolism or thrombosis of the mesenteric vessels; (11) piles, fissure, fistula.

Occult Bleeding.—This term is applied to hemorrhages of such small proportions that the blood can be detected only by chemical tests, the microscope, or the spectroscope. When all other sources of blood can be excluded (among others the ingestion of raw meat), occult bleeding is an important indication of ulcer or cancer of the digestive tract.

Watery or *serous stools* are noted in choleraic diseases, in nervous diarrhea, in the colliquative diarrhea which terminates wasting diseases, in severe enteritis, and in corrosive poisoning, as by arsenic or antimony.

Green stools may be due to the consumption of much green vegetable matter (chlorophyl); to the use of calomel or other drugs which prevent the transformation of bile-pigment into urobilin; or to diarrhea, owing to the presence of bacterial pigments or undecomposed bile-pigment.

Black stools follow intestinal hemorrhage and the use of certain drugs, as charcoal, bismuth, iron, tannin, etc.

Red stools usually indicate blood, but they may occur also after the administration of hematoxylin (logwood).

Clay-colored or *grayish-white stools* accompany obstructive jaundice. They are seen without icterus in a great variety of intestinal disorders, probably as a result of the faulty reduction of the bile-pigment. Stools may also be light colored from the presence of a large amount of fat.

Mucous stools are noted in intestinal catarrh, particularly when the lower bowel is affected, as in ileocolitis and dysentery.

Fatty stools result from the ingestion of large quantities of fats, from the absence of bile, from various diseases of the upper bowel, and from chronic pancreatic diseases.

Purulent stools result from fistula in ano, dysenteric, syphilitic, or malignant ulceration, or the rupture of abscesses into the bowel, as prostatic and pelvic abscesses.

Lienteric stools, those which contain much undigested food, are noted in inflammatory conditions of the stomach and upper bowel.

ABDOMINAL DISTENTION

Causes.—(1) Enlargement of the various organs from tumors or other causes. Recognized by the history, irregular enlargement, and special symptoms referable to the organ affected. (2) Ascites. Recognized by movable dulness with superincumbent tympany, and fluctuation. (3) Chronic peritonitis (tuberculous or cancerous) with effusion. Recognized by the history, progressive emaciation, presence of a primary lesion elsewhere, and detection of tumor-like masses, with, perhaps, pain and tenderness. (4) Tympanites. Recognized by universal tympany on percussion. (5) Pregnancy. Recognized by suppression of menses, morning emesis, pigmentation of mammary areola, softening of the cervix, intermittent uterine contractions, etc. (6) Distention of the bladder. Recognized by the history, location of dulness, and results of catheterization.

STOMATITIS

Definition.—Inflammation of the mouth.

Varieties.—The following are the most important: (1) Acute catarrhal stomatitis. (2) Aphthous stomatitis. (3) Ulcerative stomatitis. (4) Mycotic stomatitis (thrush). (5) Gangrenous stomatitis. (6) Mercurial stomatitis. (7) Gonorrheal stomatitis.

Acute Catarrhal Stomatitis.—This form is often caused by mechanical, thermic, or chemical irritation. It is a frequent accompaniment of specific fevers and wasting diseases. In children it is commonly seen in association with digestive disturbances, the result of improper food or faulty hygiene, especially of the mouth.

The chief *symptoms* are restlessness, disinclination to nurse, salivation, slight elevation of temperature, and fetor of breath, with reddening and swelling of the buccal mucosa.

Treatment.—The cause must be removed. In infants, cleansing of the mouth and of the mother's nipples, or of artificial nipples, if these be used, is imperative. Digestive disturbance should receive careful attention. Cool antiseptic mouth-washes, as a 3 per cent. solution of borax or boric acid with glycerin, are serviceable. In obstinate cases the mouth may be lightly painted with a weak solution of silver nitrate (3 or 4 grains to the ounce).

Aphthous Stomatitis.—This form is usually seen in children. Digestive disturbance is an important predisposing factor. The aphthæ begin as small vesicles, and appear in successive crops. The vesicles soon rupture and leave rounded, shallow ulcers, with yellowish bases and reddish borders. The local symptoms are those of catarrhal stomatitis. The duration of the disease is from one to two weeks.

Treatment consists in relieving the indigestion and using mild antiseptic mouth-washes. A solution of potassium permanganate (3 grains to the ounce) has been especially recommended.

Ulcerative Stomatitis.—This form of stomatitis may develop at any period of life, but it is most common in children between five and ten years of age. It is seldom seen except in debilitated subjects. As it is sometimes epidemic, some authorities regard it as contagious. The bacillus of Vincent has been found in a number of cases.

Symptoms.—The alveolar margin of the jaw is chiefly affected. The gum becomes red, swollen, and spongy. A linear ulcer, with a gray, sloughing base, soon forms, and may extend to the cheek. The glands under the jaw are swollen. In severe cases loosening of the teeth and necrosis of the bone may follow. The constitutional disturbance is often marked. Recovery, however, is the rule.

Treatment.—Hygienic conditions must be improved. Potassium chlorate is almost a specific. The dose for a child of three years is from 1 to 3 grains, in dilute solution, every three hours. The ulcers may be painted with a solution of silver nitrate (10 grains to the ounce). The official preparation of hydrogen dioxid, diluted with 3 parts of water, makes a good mouth-wash. Tonics are frequently required.

Other ulcerations of the oral mucous membrane may be traced to scurvy, metallic poisoning, infectious diseases, or to violence in cleaning the mouth (Bednar's Aphthæ).

Mycotic Stomatitis (Thrush).—This is a contagious disease of the mouth, excited by the *Saccharomyces albicans*, an organism related to the yeast fungus. It is met with in infants during the first two weeks, and in older children who have become exhausted through other diseases.

Symptoms.—The characteristic feature is the occurrence upon the mucous membrane of small milk-white elevations, which, on forcible removal, leave an abraded surface. Symptoms of catarrhal stomatitis are also present. The disease may extend to the pharynx, esophagus, and larynx. Microscopic examination reveals the mycelial threads of the fungus.

Treatment.—Generally, thrush yields to simple measures, such as removing the patches with a soft rag soaked in a solution of sodium bicarbonate (1 dram to 5 ounces) and cleansing the mouth at frequent intervals with a weak solution of borax or of sodium hyposulphite (20 grains to 1 ounce). Gastro-intestinal derangements should receive attention. To prevent reinfection, everything coming in contact with the child's mouth must be thoroughly cleansed.

Gangrenous Stomatitis (Cancrum Oris, Noma).—This comparatively rare disease usually occurs in children whose vitality has been reduced by one of the specific fevers, especially measles or whooping-cough. The spirillum of Vincent, diphtheria bacillus, or pneumococcus may be found in association with pus cocci.

Symptoms.—The cheek is the part usually affected. Externally it is swollen, hard, red, and glazed; internally there is a rapidly spreading gangrenous ulcer. The putrefaction causes an intensely fetid odor. The general symptoms are severe. The mortality is high—50 to 75 per cent. The duration is from one to two weeks, death resulting from exhaustion, bronchopneumonia, or septic diarrhea. If recovery occurs, it is usually with marked deformity from cicatrization.

Treatment.—The diseased tissue should be destroyed under anesthesia with the actual cautery or nitric acid. After the operation the mouth should be cleansed at frequent intervals with a solution of hydrogen dioxid (1:3) or of potassium permanganate (1 per cent.). Nutritious foods and stimulants are urgently needed.

Mercurial Stomatitis.—This condition may follow the continued administration of small doses of mercury at frequent intervals, the exhibition of a single large dose, or even of a single small dose, if there be an idiosyncrasy to the drug. The existence of nephritis and of cachectic states favors its occurrence.

Symptoms.—The early symptoms are a metallic taste, increase in saliva, and soreness when the teeth are brought forcibly together. The later symptoms are extreme salivation, redness, tenderness, and swelling of the gums, edema of the tongue, fetor of breath, enlargement of the submaxillary glands, and, owing to pain, great difficulty in articulation, mastication, and deglutition. In severe cases ulceration of the mucous membrane, loss of teeth, and necrosis of the jaw may result.

Treatment.—The first appearance of symptoms calls for the withdrawal of the mercury and the administration of a saline purge. The mouth may be rinsed every half hour alternately with a solution of potassium chlorate and one of hydrogen dioxid. Atropin (gr. 1/120) may be given once a day to lessen the flow of saliva, and morphin at night, if necessary, to procure sleep. Iron and other tonics may be required to combat exhaustion.

Gonorrheal stomatitis is occasionally seen in children who have been infected at birth. It is marked by catarrhal inflammation and the formation of a whitish deposit on the tongue, gums, and cheeks. It generally yields readily to applications of silver nitrate (1 to 2 per cent.).

ACUTE TONSILLITIS

Etiology.—Acute tonsillitis occurs at all ages, but it is particularly common in youth.

Exposure to cold and wet usually excites it, and such exposure is very effective when the system is debilitated or the throat is congested from improper use of the voice. In some instances the disease is epidemic and traceable to milk infection. As a secondary affection it is of frequent occurrence in acute infectious diseases, as scarlet fever, diphtheria, and variola. It is often the precursor of a systemic infection—rheumatism, chorea, septicemia, etc. Streptococci, staphylococci, diphtheria bacilli, or pneumococci may be found in the exudate.

Varieties.—(1) Follicular or lacunar. (2) Phlegmonous (quinsy).

Symptoms.—The chief symptoms are chilliness, headache and backache, high fever (103°–105° F.), pain in the throat, difficult deglutition and altered nasal voice, salivation, fetor of the breath, and swelling and tenderness behind the angles of the jaw.

In the *follicular* form the tonsils are red and swollen, and present little yellow spots on their surfaces. These spots correspond to collections of desquamated and degenerated epithelial cells in the lacunæ or crypts of the gland. During convalescence the contents of the lacunæ are often expelled in the form of cheesy pellets having a characteristic unpleasant odor.

Phlegmonous tonsillitis may be unilateral or bilateral. The affected glands are extremely swollen, sometimes almost touching one another. The pain is intense, and often radiates to the ear. The secretions of the mouth are increased. Swallowing is difficult or impossible, the voice is muffled, and breathing is embarrassed. In three or four days, however, the swollen gland softens from the formation of an abscess, and usually by the fifth or sixth day the pus is discharged spontaneously, with almost instant relief to the patient.

Complications.—Albuminuria is frequent. Endocarditis, otitis media, and a diffuse erythema occasionally occur. Suffocation from the escape of pus into the larynx and ulceration into the carotid artery are extremely rare terminations.

Diagnosis.—Follicular tonsillitis must be distinguished from scarlet fever and diphtheria.

Scarlet Fever.—The early and persistent vomiting, the very frequent pulse, the "strawberry tongue," and the peculiar punctiform eruption will suggest scarlatina.

Diphtheria.—In this disease there is an ashy-gray membrane, which cannot be readily detached, and which, if removed forcibly, leaves a bleeding surface. The membrane does not remain limited to the tonsils, but soon spreads to the pillars, uvula, and pharynx. In doubtful cases the only criterion is the presence or absence of the Klebs-Löffler bacillus.

Prognosis.—This disease is rarely fatal, and even in severe cases recovery is usually complete within ten days.

Treatment.—The patient should be confined to a warm room, and if there be much fever, to bed. A mild aperient is indicated at the outset. The diet should be light but sustaining. The sucking of ice affords relief. The most reliable internal remedies are the salicylic compounds and sodium benzoate. These should be given in full doses at frequent intervals.

℞.	Tincturæ aconiti	♏xl
	Sodii salicylatis	ʒiss
	Syrupi aurantii florum	f℥i
	Aquæ	q. s. ad f℥iij.—M.

Sig.—A dessertspoonful every two hours.

Guaiac is also recommended. A dram of the ammoniated tincture of guaiac may be given in milk every three hours. Febrile symptoms, if pronounced, may be controlled by small doses of phenacetin or by a combination of aconite and spirit of nitrous ether. The pain may be so intense as to require the use of opium.

Local Treatment.—Externally, cold applications often afford more relief than fomentations. Antiseptic sprays, like Dobell's solution (see p. 38) or a solution of hydrogen dioxid (1:4), are of decided benefit. Direct applications to the surface of the glands of finely powdered aspirin, of dry sodium bicarbonate, or of the tincture of ferric chlorid are often useful.

℞.	Potassii chloratis	gr. xx
	Tincturæ ferri chloridi	fʒiij
	Glycerini	fʒv
	Aquæ	f℥ij.—M.

Sig.—Use locally.

Scarification, followed by gargling with hot water, is another measure which frequently affords relief.

Pus should be evacuated as soon as its presence can be detected. In the majority of cases it is best to make the incision not in the tonsil itself, but in the soft palate, a little above and to the outer side of the gland.

HYPERTROPHY OF THE TONSILS

Etiology.—Hypertrophy of the tonsils occurs most frequently in childhood. While it is often excited by repeated attacks of tonsillitis, in some cases there appears to be no other cause than a congenital predisposition.

Pathology.—It may be a true hypertrophy, but in most instances overgrowth of either the glandular structure or of the connective tissue predominates, the firmness of the gland increasing in proportion to the overgrowth of the latter. The follicles are often dilated and filled with cheesy material which results from the accumulation of fatty degenerated epithelium. Nasopharyngeal catarrh, hyperplasia of the lingual tonsil, and adenoid growths in the nasopharynx are often associated conditions.

Symptoms.—The symptoms consist in mouth-breathing, snoring during sleep, dry cough, a thick voice of a nasal quality, fetor of the breath, impairment of hearing, a listless or stupid expression of countenance, mental dulness, and malnutrition. Night-terrors are common. Persistent interference with breathing through the nose gives rise to the following deformities: narrowing of the nostrils, contraction of the superior dental arch, elevation of the hard palate, and a chest conformation like that of rickets (*pigeon-breast*), or, more rarely, like that of emphysema (*barrel-chest*).

Complications.—Hypertrophy of the tonsils increases the liability to acute catarrh of the nasopharynx, to follicular tonsillitis, and to diphtheria. Chronic catarrh of the middle ear, bronchial asthma, and incontinence of urine are possible sequels.

Prognosis.—This is favorable, if proper treatment be adopted.

Treatment.—Attempts to reduce the enlargement by applying tincture of iodin, tincture of ferric chlorid, alum and glycerin, etc., usually fail. If the glands are very large and the general health is suffering, no time should be lost in resorting to tonsillectomy. Pharyngeal adenoids should also be removed.

Constitutional treatment should not be neglected. It includes systematic bathing, breathing exercises, attention to diet and clothing, and the administration of such drugs as cod-liver oil, hypophosphites, and iodid of iron.

ACUTE PHARYNGITIS

(Acute "Sore Throat;" Simple Angina)

Definition.—An acute catarrhal inflammation of the mucous membrane of the pharynx, soft palate, and uvula. It is frequently associated with tonsillitis and laryngitis.

Etiology.—Exposure to cold and wet is the most common cause. It may be of rheumatic or gouty origin. It may be excited by local irritants, such as hot drinks or the inhalation of noxious gases.

It is also met with in scarlet fever, measles, and other infectious fevers.

Symptoms.—These comprise chilliness, slight fever with its associated phenomena, stiffness and tenderness of the muscles of the neck, soreness in the throat, painful deglutition, a sensation of dryness or tickling, and a hacking cough. Extension to the larynx may cause hoarseness; to the ear, through the Eustachian tube, deafness. Inspection reveals a red and swollen mucous membrane.

Prognosis.—Favorable.

Treatment.—In mild cases a gargle of potassium chlorate will suffice. In severe cases the application to the throat of cloths wrung out of cold water proves grateful. The sucking of pieces of ice affords much relief. Gargles or sprays of the distillate of hamamelis (50 per cent.) are useful. A spray of menthol, 2 grains to the ounce of liquid petrolatum, is also efficacious. Lozenges containing cocain will often relieve pain and allay the tickling sensation in the throat. The following formula, recommended by Bosworth, answers the purpose admirably:

℞.	Cocainæ hydrochloridi	gr. v.
	Extracti krameriæ	gr. ij
	Sodii bicarbonatis	gr. xv
	Extracti glycyrrhizæ	ʒiiss.—M.

Fiant trochisci No. xxx.

Internally a mild aperient may be given at the outset. Sodium benzoate (5 grains four times daily) has a beneficial effect. Belladonna with aconite is also recommended. The rheumatic form usually yields promptly to a mild salicylic preparation like salophen or aspirin (5 to 8 grains three or four times a day).

VINCENT'S ANGINA

This is a disease affecting the tonsils and uvula, less frequently the mouth and pharynx, or lips, and characterized by the formation of a false membrane and, in marked cases, ulceration extending into the submucosa. The breath is peculiarly fetid and the corresponding lymph glands are enlarged, but the constitutional symptoms are, as a rule, comparatively mild. The average duration is from one to three weeks. Differentiation from diphtheria may be impossible without a bacteriologic examination. The disease is feebly contagious and associated with an actively motile spirillum and a fusiform bacillus. Treatment consists in the local application of tincture of iodine, salvarsan, or copper sulphate.

LUDWIG'S ANGINA

(**Angina Ludovici**)

This is a very grave and acute form of phlegmonous inflammation of the tissues about the floor of the mouth and sides of the neck. It may occur in the course of various specific fevers, or it may be excited by traumatism or carious processes at the roots of the teeth. It may end in abscess-formation or gangrene, and frequently leads to general septicemia.

CHRONIC PHARYNGITIS

Etiology.—Chronic "sore throat" may result from repeated acute attacks, from overuse or improper use of the voice, or from the prolonged action of irritants, like tobacco-smoke. It is a frequent attendant upon chronic nasal catarrh and indigestion.

Varieties.—(1) Hypertrophic; (2) atrophic.

Symptoms.—The voice is husky, and its use is followed by distress; secretion is increased, so that there is a constant desire to clear the throat; disagreeable sensations, as fulness, tickling, and the like, are frequently noted.

In the hypertrophic form (granular sore throat, clergyman's sore throat, chronic follicular pharyngitis) the mucous membrane is thick, swollen, traversed by dilated veins, and studded with numerous elevations which correspond to distended follicles and overgrown lymphatic tissue.

In the atrophic form (pharyngitis sicca) the mucous membrane is pale, smooth, glossy, and dry.

Treatment.—The removal of the cause is of prime importance. All sources of local irritation, such as misuse and overuse of the voice, mouth-breathing, excessive smoking, and intemperance in eating and drinking, must be avoided. Patients should be instructed to expel sounds by the aid of the diaphragm and abdominal muscles instead of the muscles of the throat. Nasal obstructions and adenoid growths must be removed. The habit of hawking and scraping to clear the throat should be rigidly interdicted. Digestive disturbances should receive careful attention. Tonics, like iron, strychnin, and cod-liver oil, are sometimes required.

Local Treatment.—The nasopharynx should be kept clean by frequent spraying with an antiseptic alkaline liquid, like Dobell's solution:

℞.	Sodii bicarbonatis	
	Sodii boratis . . . . āā	gr. xv
	Acidi carbolici	gr. viij
	Glycerini	fʒij
	Aquæ	f℥viij.—M.

Astringent applications are often of service; one of the following may be employed: Zinc sulphate, 5 grains to the ounce; tannin, 1 dram to the ounce of glycerin; silver nitrate, 10 to 20 grains to the ounce. In the follicular variety it is advisable to destroy the enlarged follicles by means of the galvanocautery, after which the astringent applications may be made.

RETROPHARYNGEAL ABSCESS

(Retropharyngeal Lymphadenitis)

This is a suppurative inflammation of the pharyngeal lymphatics, usually secondary to one of the specific fevers, to follicular tonsillitis, suppurative rhinitis, otitis media, or to caries of the cervical vertebræ. It occurs especially in children. It may be recognized by pain in the throat, dysphagia, dyspnea, alteration in the voice, and the detection, on inspection or palpation, of a swelling projecting from the posterior pharyngeal wall.

Treatment.—As soon as pus can be detected it should be evacuated by means of a guarded bistoury, the head of the child being held forward to prevent the escape of the pus into the larynx.

STENOSIS OF THE ESOPHAGUS

Varieties.—(1) Functional obstruction due to spasm (esophagismus). (2) Organic obstruction.

SPASM OF THE ESOPHAGUS

(Esophagismus)

Etiology.—Esophageal spasm may occur as a symptom of hydrophobia, hysteria or chorea; it may be due to the irritation of a fissure or an ulcer; or it may occur as a primary disorder in neuropathic individuals.

Symptoms.—The symptoms are difficulty in deglutition, often paroxysmal or periodic, regurgitation of food, and in some cases discomfort or actual pain during the ingestion of food. Dilatation of the esophagus may ensue.

Diagnosis.—The functional nature of the obstruction may be recognized by the neurotic associations, the marked fluctuation or periodicity in the symptoms, the slowness with which emaciation appears, and the slight or transitory resistance offered to the passage of large-sized bougies.

Treatment.—The underlying neurotic condition should receive careful attention. Bromids are sometimes useful. The systematic passage of bougies is usually followed by satisfactory results.

ORGANIC ESOPHAGEAL OBSTRUCTION

Etiology.—Stenosis of the esophagus may be due to (1) external pressure produced by an aneurysm, a tumor, enlarged glands, etc.; (2) cicatrization of an ulcer produced by corrosive acids or alkalis, syphilis, or the action of the gastric juice (peptic ulcer); (3) carcinoma of the esophageal wall.

Symptoms.—The chief symptom is slowly increasing difficulty in deglutition, with the regurgitation of food. esophagus is often much dilated above the constrict and the food may collect in the pouch thus formed, so regurgitation may be delayed for several hours. The passage of a bougie meets with a permanent obstruction. There is much loss of flesh.

Diagnosis.—The history of syphilis or of corrosive poisoning will suggest a cicatrix. Aneurysmal obstruction can usually be detected by physical examination. Aneurysm should be excluded before a bougie is passed. The age, cachexia, pain, emaciation, expectoration of blood-streaked mucus, and enlargement of the cervical glands will suggest carcinoma. The location and the degree of the stenosis are best determined by radiography or esophagoscopy.

In cicatricial stenosis and in some cases of cancerous stricture gradual dilatation with bougies is indicated. Eventually nutrient enemas and possibly gastrostomy will be demanded.

DIVERTICULA OF THE ESOPHAGUS

Two forms of diverticula—circumscribed pouch-shaped dilatations—are observed: pressure diverticula and traction diverticula. The former result from pressure within the esophagus (large boluses of food). Congenital defects may be a contributing factor. The most common location is at the junction of the pharynx and esophagus. Traction diverticula are due to the contraction of scar tissue (inflamed lymph-nodes) attached to the outer wall of the esophagus. Symptoms occur chiefly with pressure diverticula and consist of dysphagia and regurgitation of food. The diagnosis is best made by radiographic examination.

ACUTE GASTRITIS

(Acute Gastric Catarrh)

Etiology.—As a primary condition, acute gastritis may result from the eating of indigestible food, partially decomposed food or excessive quantities of food; or it may follow the ingestion of chemical irritants—alcohol, strong acids or alkalis, corrosive sublimate, etc. As a secondary condition it is present in many of the acute infectious diseases.

Pathology.—The mucous membrane is red, swollen, and covered with thick mucus. It is sometimes the seat of ecchymosis. The microscopic changes consist in marked mucoid degeneration and cloudy swelling of the epithelial cells, and the infiltration of the interstitial tissues with round cells.

In toxic gastritis there is often extensive sloughing of the gastric mucosa.

Symptoms.—The symptoms vary much in degree. In mild cases there are anorexia, a feeling of discomfort and fulness, eructations, nausea, and, perhaps, vomiting. The tongue is heavily coated. In severe cases the symptoms are more marked, particularly the nausea and vomiting. There may be also moderate fever (102°–103° F.), thirst, herpes, distention of the epigastrium, local tenderness, and considerable prostration. The vomitus is composed at first of sour, fermented food; later, of mucus and bile. Jaundice may follow from the extension of the catarrh to the duodenum and bile-ducts, and diarrhea from its extension to the intestines.

Toxic gastritis is manifested by intense burning pain in the throat, gullet, and stomach, persistent vomiting of food-remnants mixed with blood and mucus, marked abdominal tenderness, and the phenomena of collapse. Atrophy of the mucosa and cicatricial stenosis of the orifices are common sequels in cases that do not prove immediately fatal.

Diagnosis.—It may resemble the onset of *scarlet fever*, but the history of contagion, the "strawberry tongue," sore throat, very rapid pulse, and eruption will lead to the recognition of the latter.

Prognosis.—Simple acute gastritis usually runs a favorable course, and rarely lasts more than a few days.

Treatment.—If the stomach has not been completely emptied, an emetic, such as warm water or ipecac, should be employed. Locally, a mustard-plaster or a turpentine stupe will aid in relieving distress. As a rule, no food should be given by the mouth until the stomach becomes retentive. Ice, however, may be allowed to quench the thirst. In delicate subjects nutrient enemata will be required. If there is constipation, a mercurial laxative may be given with advantage. Such a combination as the following usually acts favorably:

℞.	Hydrargyri chloridi mitis	gr. j
	Bismuthi subnitratis	gr. xx.—M.

Fiant chartulæ No. vj.

SIG.—One on the tongue every hour, to be followed by a Seidlitz powder, if necessary.

Severe pain, nausea, restlessness, and insomnia are best relieved by opium suppositories. Persistent vomiting may be relieved by bismuth subnitrate (10 grains) combined with creosote (½ minim), with cocain (⅙ grain), or with hydrocyanic acid, as in the following formula:

℞.	Bismuthi subnitratis	ʒiij
	Acidi hydrocyanici diluti	♏xxxij
	Aquæ	f℥iv.—M.

SIG.—Shake well. A dessertspoonful every three hours.

The following combination of ipecac and nux vomica is often serviceable:

℞.	Tincturæ nucis vomicæ Vini ipecacuanhæ	āā f℥ij.—M.

SIG.—Two drops every hour.

After the lapse of twenty-four or thirty-six hours it is usually possible to give bland nourishment by the mouth. Barley-water, champagne with soda-water, milk and lime-water, peptonized milk, and light broths may be given in small quantities at frequent intervals. The return to solid food should always be carried out very gradually.

The treatment of *toxic gastritis* consists in the immediate neutralization of the poison by chemical antidotes, in the evacuation of the stomach (except in the late stages of poisoning by caustics) by the stomach-pump or emetics, and in the administration of demulcents and opium.

CHRONIC GASTRITIS

(Chronic Gastric Catarrh; Catarrhal Dyspepsia)

Etiology.—It may be excited—(1) By prolonged irritation of the stomach, such as results from errors in diet (excesses in eating and drinking, indigestible food, irregular meals, deficient mastication, etc.), or from the excessive use of alcohol, tobacco, condiments, or purgatives; (2) by passive congestion the result of chronic heart disease or cirrhosis of the liver; (3) by chronic diseases that disturb metabolism, such as tuberculosis, diabetes, chronic Bright's disease, gout, chlorosis, etc.; (4) by chronic diseases of the stomach itself, such as cancer, ulcer, gastrectasia, etc.

Pathology.—The mucous membrane is of a grayish or slaty color, swollen, and covered with tenacious mucus. The veins are dilated, and there may be ecchymoses. Microscopically, there is a cellular infiltration in the interstitial tissue. The glands are dilated, elongated, and tortuous, and their epithelium is more or less degenerated and detached. The interglandular proliferation may be so pronounced as to cause great thickening of the mucous membrane (*hypertrophic gastritis*), or, on the other hand, the new-formed fibrous tissue may contract to such a degree as to cause extreme thinning of the coats of the stomach and atrophy or complete destruction of the glandular elements (*atrophic gastritis*).

Symptoms.—The *subjective symptoms* are very variable, and, for the most part, not characteristic. The chief phenomena are furring of the tongue, fetor of the breath, anorexia, discomfort in the epigastrium, especially after eating solids, belching, eructations, heartburn, constipation, headache, vertigo, and attacks of palpitation. Nausea and vomiting are not uncommon. The latter may occur before breakfast or at the height of digestion. If it occurs on rising in the morning, the vomit consists of frothy mucus; if it occurs after meals, the vomit is composed of undigested food remnants intimately mixed with more or less ropy mucus. The entire epigastrium may be sensitive to pressure.

Examination of the gastric contents reveals an excessive amount of mucus containing many leukocytes, and usually diminished secretion of hydrochloric acid, but not infrequently the acidity is normal or increased (*gastritis acida*). In complicated cases there is no motor insufficiency.

Chronic gastric catarrh rarely terminates in *atrophic gastritis* (*achylia gastrica*), the most important symptoms of which are paroxysmal pain, more or less persistent vomiting, constipation alternating with diarrhea, emaciation, severe anemia, and absence of free HCl and of digestive ferments from the stomach-contents after a test-breakfast.

Diagnosis.—*Atony of the Stomach.*—In simple atony fluids excite as much distress as solids, vomiting is rare, the secretion of mucus is not increased, the secretion of HCl is not usually decreased, and considerable quantities of undigested food can be recovered from the stomach seven hours after a test-meal.

Hyperchlorhydria.—In this condition the general health is not impaired, the appetite is usually good, there is more or less severe pain shortly after eating, albumins and alkalis relieve the pain, and excess of HCl is found in the stomach-contents; albumin-digestion is good, starch-digestion is retarded, and there is no excess of mucus.

Nervous Dyspepsia.—In this syndrome the severity of the symptoms varies considerably from day to day according to the mental state of the patient, and is not materially influenced by the quantity or the quality of the food; the general health is not often impaired, the nervous symptoms are very prominent, the secretion of the stomach is usually normal, and there is no excess of mucus.

Peptic Ulcer.—The severe, localized paroxysms of pain shortly after eating, the localized tenderness, hematemesis, and hyperacidity will serve to distinguish ulcer from catarrh.

Cancer of the Stomach.—The history, rapid course, cachexia, persistent vomiting, hematemesis, palpable tumor, signs of gastrectasis, and the early absence of free HCl from the gastric juice, with the presence of large quantities of lactic acid and of the Boas-Oppler bacilli, will usually render the diagnosis clear.

Care must be taken to determine whether the catarrh is *primary* or *secondary* to some constitutional or visceral disease.

Prognosis.—The primary forms of chronic gastritis, if not too far advanced, are frequently cured. The prognosis is unfavorable when there is much atrophy of the gastric mucosa. In the secondary forms the prognosis is dependent on that of the primary disease.

Treatment.—The cause should be ascertained and removed if possible. Regularity in the time of meals, slowness in eating, and thorough mastication of food must be insisted upon. The patient should be cautioned against overeating and the taking of large quantities of liquid, especially of iced water, during meals. Overindulgence in alcohol, tobacco, coffee, and tea should be forbidden. The resumption of mental or physical work immediately after meals should also be avoided.

A mixed diet of bland, readily digestible food is required. It may usually include boiled, baked, or grilled beef and mutton, chicken, sweetbread, boiled fish, oysters, soft-boiled or poached eggs, pulled bread, fresh butter, baked potato, young string-beans, small peas, spinach, hearts of celery, thoroughly cooked cereals, calf's-foot jelly, and junket. Tea, coffee, and cocoa may or may not be permissible.

An exclusive milk diet acts exceedingly well in some cases. Systematic lavage is of great value in severe cases, especially when there is excessive secretion of mucus. When lavage cannot be tolerated, the stomach may be cleansed by a glass of hot alkaline water slowly sipped a half-hour or more before breakfast. The following artificial Carlsbad salt may be used as the alkali:

℞. Sodii sulphatis	ʒx
Sodii bicarbonatis	ʒiv
Sodii chloridi	ʒij.—M.

Sig.—A teaspoonful in a glass of hot water an hour before breakfast.

In mild cases the administration of a bitter—calumba, gentian, nux vomica—some time before meals often proves efficacious. In many cases an alkali may be added with advantage to the bitter, as in the following formula:

℞. Sodii bicarbonatis	ʒiss
Infusi gentianæ compositi	f℥vj.—M.

Sig.—A tablespoonful before meals.

When the stomach is highly sensitive, silver nitrate will be found a valuable remedy. It may be given in pill form in combination with hyoscyamus, as in the following formula:

℞. Argenti nitratis	gr. vj
Extracti hyoscyami	gr. x.—M.

Fiant pilulæ No. xx.

Sig.—One pill a half-hour before meals.

Bismuth subnitrate is also of service in such cases. Diluted hydrochloric acid is sometimes serviceable in replacing the natural acid of the gastric juice. In many cases, however, better results are secured from the administration, during meals, of pancreatin with sodium bicarbonate. Flatulence and fermentation may sometimes be controlled by such antiseptics as bismuth salicylate, creosote, bismuth-beta-naphthol, etc. The following combination is often of value:

℞. Creosoti	♏xx
Bismuth-beta-naphthol	gr. c
Pulveris zingiberis	gr. xxx.—M.

Pone in capsulas No. xx.

Sig.—One after meals.

So far as possible, constipation should be overcome by regulation of diet, systematic exercise, and the use of enemas or suppositories.

Change of scene, a sunny climate, good hours, and freedom from business worry and household cares often prove more beneficial than any other measure employed.

ATONY OF THE STOMACH

(Motor Insufficiency; Myasthenia Gastrica)

Definition.—Atony of the stomach consists in relaxation of the muscular coat of the stomach and insufficiency of its propulsive powers. It frequently leads to gastrectasis.

Etiology.—Motor insufficiency is of common occurrence. It may be congenital; it may be caused by intemperance in eating and drinking; it may follow acute infections; it may occur in the course of chronic diseases attended by malnutrition; it may appear acutely after traumatism or intense emotional excitement; it may be a complication in other diseases of the stomach, especially in gastroptosis, chronic gastritis, nervous dyspepsia, and hypersecretion.

Symptoms.—In simple atony the chief symptoms are a feeling of fulness and discomfort after meals, especially if the latter have been large, and frequent belching of gas. The severity of the symptoms often bears a definite relation to the quantity of food taken. Fluids are as likely to excite distress as solids. As a rule, there is neither vomiting nor pain. The appetite is usually good, the general health is not seriously affected, and the symptoms entirely abate upon the evacuation of the stomach. There are no signs of gastrectasia. When the intestines are similarly affected, there may be marked nervous symptoms—headache, vertigo, and paresthesia—and considerable disturbance of nutrition.

An exact diagnosis rests upon the examination of the gastric contents. No remnants of food can be obtained from the stomach in the morning before breakfast, but a considerable quantity of solid matter can be recovered from the stomach one hour after the ingestion of the Ewald test-breakfast, or seven hours after the ingestion of the Riegel test-dinner.

Prognosis.—This is favorable, if the cause can be removed.

Treatment.—The first indication is to remove the cause. The food should be readily digestible, small in bulk, finely divided, and nutritious. Fluids, except in moderate quantities, and coarse vegetables are to be avoided. The diet may include tender meats, eggs, oysters, boiled fish, well-cooked cereals, steamed rice, stale bread, fresh butter, baked potatoes, tender spinach, string-beans, and asparagus-tips. It is rarely necessary to increase the number of meals. Rest for at least an hour after large meals is to be urged. Exercise in the open air and frequent tepid baths are general measures of value. Lavage is unnecessary unless there are gastrectasis and fermentation.

General tonics, especially iron, are often needed. The most useful direct remedies are the bitters (quassia, gentian, and calumba), particularly the tincture of nux vomica, which may be given in doses of from 5 to 10 minims, gradually increased, before meals. Alkalis are indicated when there is excessive acidity. Antifermentatives, particularly small doses of creosote, phenol, or betanaphthol, are useful in reducing flatulence.

Constipation is best relieved by diet, abdominal massage, and enemas.

NERVOUS DYSPEPSIA

(Neurasthenia Gastrica)

Definition.—The characteristic feature of this syndrome is pronounced discomfort during the period of digestion, out of all proportion to the disturbances of gastric secretion or motility. The source of the discomfort appears to be an excessive irritability of the nerves of the stomach.

Etiology.—Nervous dyspepsia usually occurs in those of a distinctly nervous temperament, and mental overexertion, worry, and excesses are potent etiologic factors. It is frequently associated with neurasthenia and hysteria. It may be due to reflex irritation from other organs.

Symptoms.—The tongue is often clean. The appetite is very variable—at one time it is lost, at another it is inordinate, at another it is perverted, the patient craving unusual articles for food. Pain during the period of digestion is a prominent symptom. It varies in intensity from a feeling of discomfort to the most violent distress. There is rarely tenderness, but the skin over the stomach is often abnormally sensitive.

Belching is common. Vomiting is not frequent. Exaggerated peristaltic movements attended with gurgling sounds (peristaltic unrest) may be perceptible to the patient. Nervous phenomena—headache, vertigo, disturbed sleep, hypochondriasis, lassitude, and palpitation—are conspicuous.

Gastric acidity is usually normal, but there may be subacidity or hyperacidity. In the majority of cases gastric motility is not affected, the viscus emptying itself within the normal period.

The symptoms are usually confined to the period of digestion; they are out of proportion to the disturbance of the digestive functions; they vary greatly from day to day, according to the mood of the patient; and they are not materially influenced by the quality or the quantity of the food.

Prognosis.—This is good, if the cause can be removed.

Treatment.—The treatment is largely that of neurasthenia. The avoidance of excitement and of excessive mental work must be enjoined. An extended voyage may effect a cure. In brain-workers systematic exercise in the open air and frequent bathing, followed by friction of the skin, often prove very efficacious. On the other hand, the exhausted and anemic may demand the "rest-cure." The diet should be bland and readily digestible. In many cases milk is an appropriate food. Tonics, like iron and arsenic, are often indicated. Short courses of an unirritating bromid, like that of strontium, sometimes do good. The following combination of antispasmodics is useful in certain cases:

℞. Zinci valeratis	gr. xxx
Extracti sumbul	gr. xx
Arseni trioxidi	gr. ⅓
Extracti gentianæ	gr. x.—M

Fiant pilulæ No. xx.
Sig.—One pill after each meal.

HYPERCHLORHYDRIA

(Superacidity; Hyperacidity)

Definition.—These terms are used to designate an abnormal increase in the secretion of hydrochloric acid during the digestive act. Symptoms are not likely to arise from hyperacidity unless there is also excessive irritability of the gastric mucosa.

Etiology.—This anomaly of secretion is most frequently seen in nervous individuals between the ages of fifteen and forty. Mental overexertion, the excessive use of tobacco, overindulgence in condiments, and insufficient mastication are important predisposing factors. It is a common complication in chlorosis, in gastric ulcer, and in cholelithiasis.

Symptoms.—The symptoms do not appear immediately after eating, but at the acme of digestion, and include sensory irritation, varying in degree from slight discomfort to agonizing pain, with acid eructations, heartburn, thirst, diffuse tenderness over the stomach, and occasionally vomiting. These symptoms are relieved by eating small quantities of albuminous food and by the ingestion of alkalis, and disappear spontaneously upon evacuation of the stomach. They may be continuous or periodic.

The stomach-contents obtained after a test-breakfast are excessively rich in hydrochloric acid, both free and combined. Albumin digestion is rapid. The resting stomach is empty. There is no motor insufficiency.

Prognosis.—In the absence of complications the prognosis is favorable.

Treatment.—The cause should be ascertained and removed, if possible. Thorough mastication is imperative. The diet should be unirritating, and composed of the more digestible meats, farinaceous foods, and vegetables. Coarse substances, vinegar, spices, condiments, coffee, and alcohol should be avoided. A moderate amount of water or weak tea at meals is desirable. Fats, in the form of cream and butter, are usually well borne. It is sometimes desirable to increase the number of meals.

Alkalis, in the form of sodium bicarbonate or magnesia, administered at the height of digestion, relieve the symptoms. Silver nitrate (1/4 grain) with extract of belladonna (1/8 grain) thrice daily, on an empty stomach, is useful. Silver nitrate (1:2000 to 1:1000) may also be given as a stomach-douche with advantage. As there is often marked irritability of the gastric mucosa in these cases, sedatives, like bromids, valerates, and sumbul, are sometimes of service.

GASTROSUCCORRHEA

(Reichmann's Disease)

Definition.—This is a functional condition characterized by the secretion of large quantities of gastric juice, even when the stomach is empty. It is often associated with hyperchlorhydria. Two forms have been recognized—(1) the continuous and (2) the intermittent.

Etiology.—The causes of gastrosuccorrhea are the same as those which excite hyperchlorhydria.

Symptoms.—In the continuous form the symptoms appear regularly, but with varying intensity, and consist in more or less severe pain, both at the acme of digestion and in the night; vomiting of large quantities of yellowish, acid fluid, even when the ingesta are no longer in the stomach; marked thirst; acid eructations; and headache, sometimes of a migrainous type. The ingestion of a small quantity of albuminous food usually relieves the pain. Albumin digestion is good, but starch digestion is retarded. The diagnosis is rendered certain by the finding of from 50 c.c. to 500 c.c. or more of gastric juice, *without any admixture of food*, in the stomach before breakfast, particularly if lavage has been practised the night before.

Complications.—Gastrectasis may result from imperfect digestion of starches or from spasm of the pylorus excited by excessive acidity. Ulcer may coexist. In rare instances tetany develops.

Diagnosis.—In *hyperchlorhydria* the resting stomach is empty and pain does not occur at night. Care must be taken to exclude *locomotor ataxia*, of which intermittent gastrosuccorrhea may be an early symptom.

Prognosis.—This is guardedly favorable in uncomplicated cases. Relapses are common.

Treatment.—This is much the same as that for hyperchlorhydria. The painful attacks may be relieved by the administration of alkalis, or, better, by thorough lavage. Belladonna appears to possess some power to reduce gastric secretion.

GASTRALGIA

(Gastrodynia; Neuralgia of the Stomach)

Definition—Violent paroxysmal gastric pain, occurring independently of any organic disease of the stomach and of any disturbances of secretion or motility. It is a very rare affection.

Etiology.—It is more common in women than in men. Overwork, worry, sexual excesses, abuse of tobacco, reflex irritation, and anemia predispose to it. It may be a symptom of neurasthenia.

Symptoms.—The characteristic features are paroxysms of intense pain, occurring suddenly at irregular intervals, radiating to the chest and back, bearing no definite relation to eating, and lasting from a few minutes to several hours. Vomiting is rare. Pressure over the stomach may relieve the pain, and so may the taking of food.

Diagnosis.—Idiopathic gastralgia must be separated from the paroxysmal pain that occurs in gastric ulcer, gastric cancer, hyperchlorhydria, locomotor ataxia, angina pectoris, and in renal and biliary colic.

Gastric Ulcer.—Pain is excited by food and digestion, disappears upon evacuation of the stomach, is associated with hyperacidity, and often with vomiting, hematemesis, and local tenderness.

Gastric Cancer.—The pain is usually more or less continuous, and is aggravated by digestion. There may be persistent vomiting, hematemesis, cachexia, inacidity with lactic-acid fermentation, and a palpable tumor.

Hyperchlorhydria.—The pain is digestive, and is relieved by alkalis and by albuminous food. Examination of the stomach-contents reveals excess of HCl.

Crises of Tabes.—Unsteadiness of gait and of station, Argyll-Robertson pupil, shooting pains in the limbs, abnormalities of sensation, and abolition of deep reflexes will indicate locomotor ataxia.

Angina Pectoris.—The pain radiates from the heart to the neck and arm, is frequently excited by exertion or indiscretions in diet, is generally of short duration, is often attended with immobility of the body and a feeling of imminent dissolution, and is usually associated with the signs of arteriosclerosis.

Renal Colic.—The pain radiates from the kidney into the ureter of the affected side, and concretions or blood may be found in the urine.

Biliary Colic.—The pain is usually in the right hypochondriac region, and is often accompanied by chill, fever, and jaundice. The liver and gall-bladder may be enlarged and tender.

Prognosis.—This is favorable in uncomplicated cases.

Treatment.—*The Attack.*—Hot applications are useful. Galvanization (the anode over the stomach and the cathode near the spinal column) often affords prompt relief. The most generally efficacious remedies are antipyrin (8 grains), brandy (1 to 2 fluidrams), aromatic spirit of ammonia (½ fluidram), chloroform (2 to 5 minims), and diluted hydrocyanic acid (2 minims). These remedies are most efficacious when given in hot water. Such a combination as the following is frequently successful:

℞. Chloroformi . . . . f ʒ iss
Spiritus ammoniæ aromatici
Spiritus vini gallici
Tincturæ cardamomi compositæ . . āā f ʒ v.—M.
Sig.—A teaspoonful in hot water every fifteen or thirty minutes.

In very severe cases it will be necessary to resort to morphin.

The Interval.—The cause must be ascertained, and, if possible, removed. The habits of the patient must be corrected. Methods of treatment intended to improve the general nutrition are of the greatest value. When there is anemia, iron will be found very useful. Among special remedies arsenic, valerianates, sumbul, quinin, and cannabis indica are available. The following combination often proves efficacious:

℞. Arseni trioxidi . . . . gr. ⅕
Quininæ valeratis . . . . gr. xxx
Ferri pyrophosphatis . . . . . .
Extracti sumbul . . . . āā gr. xx.—M.
Fiant in pilulæ No. xx.
Sig.—One pill after each meal.

In some cases a complete change of scene or enforced rest in bed for a given period is the only means of effecting a cure.

PEPTIC ULCER OF THE STOMACH AND DUODENUM

(Round or Perforating Ulcer of the Stomach and Duodenum)

Definition.—A circumscribed loss of tissue in the stomach or duodenum, usually involving both the mucous membrane and the deeper structures, and characterized clinically by paroxysmal pain, localized tenderness, vomiting, hematemesis, and hyperacidity of the gastric juice.

Similar lesions occasionally occur at the lower end of the esophagus, and after gastro-enterostomy in the jejunum.

Etiology.—It is more common in women than in men. The majority of cases occur between the ages of twenty and forty. Chlorosis and anemia are important predisposing factors.

Duodenal ulcer not infrequently follows large superficial burns.

Pathogenesis.—It is generally admitted that these ulcers are due to the digestive action of the gastric juice upon an area of local malnutrition. The cause of the malnutrition is obscure. It has been ascribed to thrombosis or embolism, to bacterial infection, to the elimination of toxins by the gastric or duodenal mucosa and to excessive vagal irritability causing spasmodic muscular contraction.

Pathology.—As a rule, the ulcers are single, but they may be multiple. The most frequent seat in the stomach is the posterior wall, in the lesser curvature, near the pylorus; in the duodenum, in the ascending portion about half an inch from the pylorus. They have a punched-out appearance, are round or oval in outline, and, if recent, are funnel-shaped, with the apex toward the serous coat. The edges are usually smooth, rarely ragged. They vary in diameter from a few millimeters to several centimeters, and may extend to the muscularis or even to the serosa.

Symptoms.—Symptoms of indigestion are generally present. The characteristic symptoms are:

1. *Pain.*—This is usually paroxysmal, severe, and localized. It may radiate to the back or sides. In many cases the pain is induced or aggravated by taking food, and continues until the stomach-contents are removed either by vomiting or by spontaneous discharge into the intestine. In other cases especially of duodenal ulcer, the pain comes or when the stomach is empty, that is, midway between meals or in the early morning hours (hunger pain) and is relieved by bland food. Very often the attacks of pain occur at intervals of weeks or months.

2. *Localized Tenderness.*—Two small areas of tenderness

can often be elicited, one in front below the ensiform cartilage, and one behind, a little to the left of the tenth or eleventh dorsal vertebra.

3. *Vomiting.*—This frequently occurs in from one-half hour to two hours after eating. The vomit usually consists of undigested food and acid fluid.

4. *Hematemesis.* —This occurs in more than one-half of all cases. It is the cause of death in about 20 per cent. of all fatal cases of ulcer. The blood is generally fluid and unaltered, but if retained in the stomach for some time, it may have a coffee-ground appearance. In some cases the blood is discharged entirely by the bowel.

Not rarely the bleeding is "occult," or invisible to the naked eye, but may be detected by the benzidin test.

5. *Hyperacidity.*—An increase of HCl is noted after a test-meal in the majority of cases. The acidity, however, may be normal.

In some cases only the symptoms of dyspepsia are present, while in others all symptoms are absent, the disease passing unrecognized until sudden perforation or profuse hemorrhage occurs. Usually the perforation is into the peritoneum, but very rarely it is into the pleura, pericardium, or intestines.

Sequels.—(1) Perforation. This occurs in from 8 to 10 per cent. of all cases. (2) General or circumscribed peritonitis. General peritonitis is usually the result of perforation; circumscribed productive peritonitis is a conservative process and results from the direct extension of the inflammatory process through the stomach-wall. It gives rise to perigastric adhesions. (3) Subphrenic abscesses. This is usually the result of perforation after the formation of adhesions. (4) Stenosis of the pylorus, stenosis of the cardia, or hour-glass constriction of the stomach may result from the contraction of cicatrices. (5) Cancer not infrequently develops on the basis of an old ulcer.

Diagnosis.—*Hyperchlorhydria.*—In this condition the pain does not occur so regularly nor so soon after eating; it is not modified by position, but is often completely relieved by eating albuminous food. Hematemesis is absent and there are no tender spots.

Gastralgia.—In this affection the pain occurs at irregular intervals, is not dependent upon eating (often occurring when the stomach is empty), is relieved by pressure, and is not associated with tender spots, hematemesis, or hyperacidity.

Cancer of the Stomach.—The history, rapid course, advanced cachexia, palpable tumor, vomiting of large quantities of undigested food at long and irregular intervals, "coffee-ground vomit," abundance of lactic acid with Boas-Oppler bacilli, and the absence of free hydrochloric acid will point to cancer.

Duodenal Ulcer.—In this lesion the pain is further to the right and occurs, as a rule, later after the meals, the blood is usually evacuated through the bowel, and there is little or no vomiting.

Cholelithiasis.—In this condition the pains appear more suddenly, occur at more irregular intervals, and often independently of eating, usually radiate toward the right shoulder, and are often associated with swelling and tenderness of the liver, enlargement of the gall-bladder, slight fever, chill, and jaundice.

Prognosis.—This is guardedly favorable in recent cases. The mortality in all cases is from 8 to 10 per cent. Some ulcers run a rapid course and end fatally through hemorrhage or perforation; others, even without treatment, persist for many years. Relapses are common.

Treatment.—Rest and appropriate diet are the most important factors in the treatment. The rest should be kept up for from six to twelve weeks, and for the first two or three weeks of this period the patient should be confined to bed. If hemorrhage has recently occurred or if vomiting be urgent, it is advisable to withhold all food from the stomach for a few days and to nourish the patient by means of nutritive or saline enemas.

After the pain and vomiting have sensibly abated, feeding by the mouth should be resumed. The diet should consist of milk, buttermilk, beef-juice, animal broths, egg-white, and thin pap. As soon as the gastric symptoms have completely disappeared, which will rarely be before the lapse of three or four weeks, the patient may be allowed such articles as soft-boiled eggs, scraped beef, boiled sweet-breads, the tender part of oysters, white meat of chicken, well-made gruel, and custard pudding.

The most useful drugs are alkalis, silver nitrate, and bismuth subnitrate. The alkalis are useful in overcoming the superacidity of the gastric juice. Sodium bicarbonate is one of the best; it may be combined with magnesia or chalk, according as there is constipation or diarrhea. Artificial Carlsbad salt (see p. 45) is an excellent alkaline laxative; of this, a teaspoonful or more may be given in a half pint of hot water in the early morning. Silver nitrate and bismuth subnitrate are valuable remedies and may be used alternately, each for a period of a week or ten days. They may be prescribed as follows:

℞. Argenti nitratis
Extracti belladonnæ . . . . . . āā gr. vj.—M.
Fiant pilulæ No. xx.
Sig.—One pill half an hour before meals.

℞. Bismuthi subnitratis . . . . ʒvj
Acidi hydrocyanici diluti . . . . . . ♏xxiv
Aquæ . . . . f℥vj.—M.
Sig.—A tablespoonful three or four times, half an hour before meals.

Pain and vomiting usually yield to complete rest, rectal feeding, and the administration of silver nitrate or bismuth subnitrate. In some cases it may be necessary to use morphin hypodermically. Externally, stupes or sinapisms are sometimes useful. The treatment of hematemesis is considered on page 67.

Surgical Treatment.—In all cases of perforation an operation should be done at the earliest possible moment. If life is threatened by repeated hemorrhage, operation in the interval between the attacks offers the best method of relief. Again, an operation (gastro-enterostomy or partial gastrectomy) is indicated if there is evidence of pyloric obstruction, or of hour-glass contraction or other serious deformity of the stomach, or if the disease does not yield to medical treatment and the life of the patient is endangered by malnutrition.

CANCER OF THE STOMACH

Etiology.—*Sex.*—Cancer of the stomach is somewhat more common in men than in women.

Age.—The majority of cases occur between the ages of forty and sixty. It is rare before thirty.

Heredity.—About 8 per cent. of the cases appear to be hereditary.

Prolonged Irritation.—Cancer sometimes develops on the basis of an old ulcer.

Pathology.—Cancer of the stomach is almost always primary. The pylorus is the part most frequently attacked. After the pylorus the points of predilection are the lesser curvature and cardia. The following varieties are encountered: Scirrhus or hard cancer, medullary or soft cancer, adenocarcinoma (cylindric-celled epithelioma), colloid cancer, and squamous-celled epithelioma. Ulceration is rare in scirrhus, but common in medullary cancer and adenocarcinoma. Colloid cancer appears most commonly as a diffuse infiltration of the stomach-wall. Squamous-celled cancer is rare, and occurs only at the cardia.

Owing to stenosis of the pylorus the stomach is usually dilated. Stagnation of the stomach-contents and the absence of hydrochloric-acid secretion favor the development of lactic-acid fermentation.

Symptoms.—Symptoms of dyspepsia are usually present. The characteristic phenomena are:

1. *Pain.*—This is rarely intense; though aggravated by

eating, it is often more or less continuous. It may radiate to the back.

2. *Vomiting.*—This is very common. When the pylorus is obstructed, the vomiting is persistent and occurs long after eating, sometimes at intervals of several days. The vomit is frequently large in amount, and is composed chiefly of undigested food and turbid fluid. It very rarely contains sarcinæ, but long, thread-like bacilli (*Boas-Oppler bacilli*) are almost constantly present and possess some diagnostic significance.

3. *Hematemesis.*—As the bleeding is slight and the blood remains for some time in the stomach, the vomit in many cases acquires a coffee-ground appearance. For a long time the bleeding may be "occult."

4. *Enterorrhagia.*—Blood in the stools, often only recognizable by chemical tests, is present in a large percentage of cases.

5. *Cachexia.*—The anemia, weåkness, and emaciation are often disproportionate to the loss of nourishment.

6. *Palpable Tumor.*—A movable, tender mass can be detected sooner or later in a large proportion of all cases.

7. *Absence of HCl with Lactic-acid Fermentation.*—The absence of free HCl and the presence of large quantities of lactic aid, while not peculiar to cancer, are strongly indicative of the disease.

In addition to these features the symptoms and signs of *gastrectasis* are frequently present.

Complications and Sequels.—Metastases in neighboring structures—liver, lymph-glands, pancreas, and peritoneum—are of common occurrence. Ascites and edema are occasionally encountered. Perforation, subphrenic abscess, tetany, venous thrombosis, multiple neuritis, and coma (from β-oxybutyric-acid intoxication) are rare complications.

Diagnosis.—The differential points between cancer and ulcer and cancer and chronic gastritis have already been considered.

Prognosis.—The disease is almost invariably fatal. The average duration of life is from one to two years. Marked temporary improvement frequently occurs under treatment and may prove very misleading.

Treatment.—In the early stages of the disease, when the pylorus is still patulous, a mixed diet of readily digested food is often well borne. Later, when there is retention, food should be selected that will make small demands on the stomach and that will leave little residue. Bitters—calumba, gentian, condurango—are sometimes employed with advantage. In many cases, but by no means invariably, hydrochloric acid and pepsin are useful. Lavage affords the best means of relieving the distressing symptoms resulting from retention. Vomiting not dependent upon retention may be treated with such remedies as carbonated water, hydrocyanic acid, creosote, cerium oxalate, and bismuth subnitrate. In obstinate cases rectal feeding may be required for a time. Acid eructations and flatulency are sometimes relieved by antacids and internal antiseptics, but generally lavage is much more effective. Pain will require opium, sedatives like hydrocyanic acid or chloroform, and hot applications.

Early operative interference may prolong life for several months or several years.

DILATATION OF THE STOMACH

(Gastrectasis)

Etiology.—Chronic gastrectasis may result from—(1) Atony of the stomach-walls (see p. 47); (2) from stenosis of the pylorus.

Stenosis of the pylorus may be caused by—(*a*) Congenital stricture; (*b*) carcinoma of the pylorus; (*c*) cicatrix from ulcer; (*d*) hypertrophy of the pylorus from gastric catarrh or pylorospasm excited by hypersecretion; (*e*) pressure from without, as by tumors, adhesions, floating kidney, etc.

Pathology.—All degrees of dilatation are encountered. The most severe forms are usually the result of pyloric stenosis. In atonic dilatation the stomach-walls are thin and atrophic; in dilatation from obstruction there may be marked muscular hypertrophy at the pyloric end.

Symptoms.—These vary with the cause and the degree of dilatation. In well-marked cases the chief symptoms are a feeling of fulness and discomfort after meals, frequent belching and acid eructations, increased thirst, constipation, deficient urination, and more or less emaciation. Owing to reflex irritation or autointoxication nervous symptoms often develop.

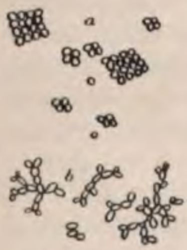

FIG. 1.—*a*, Sarcinæ ventriculi. *b*, Torculæ cerevisiæ.

Vomiting is a characteristic symptom, especially when there is stenosis of the pylorus. It occurs long after meals, sometimes at intervals of several days. The vomit is often excessive in amount, is sour and fermented, and on standing separates into a sediment of undigested food and a supernatant liquid, which is turbid and frothy. Not infrequently the vomit contains remnants of food that was eaten several days before. Microscopic examination may reveal, in atonic dilatation, numerous yeast-cells and sarcinæ, and in cancerous dilatation, the thread-like bacilli of Oppler.

PHYSICAL SIGNS.—*Inspection.*—The abdomen may be unduly prominent. In some cases the outlines of the enlarged stomach are distinctly visible. Peristaltic waves extending from left to right are frequently seen, especially in stenotic dilatation.

Palpation.—When the stomach-walls are sufficiently tense, the boundaries of the organ may be determined by palpation. In many cases of obstructive dilatation a tumor can be felt at the pylorus.

Percussion.—When the stomach contains fluid, an area dulness is found at the level of the umbilicus, or below it when the patient is erect, but not when he is lying down. This area of dulness also disappears upon the complete evacuation of the stomach.

After artificial inflation of the stomach with air or carbonic-acid gas percussion reveals an increased area of gastric tympany.

Auscultation.—The detection of splashing sounds over the stomach in the morning before breakfast or seven hours after a Riegel test-meal points to dilatation.

Mensuration.—Normally a rigid sound can be inserted a distance of 24 inches (60 cm.) from the incisor teeth; in dilatation it may be inserted as much as 28 inches (70 cm.). As the depth to which the sound can be passed is frequently increased also in gastroptosis, this sign is unreliable.

X-rays.—Skiagraphs taken after the ingestion of large quantities of bismuth subcarbonate suspended in buttermilk or porridge have proved of great value in the diagnosis of gastrectasis.

Examination of the Stomach-contents.—The detection of remnants of food in the stomach in the morning after a simple supper renders the diagnosis of severe motor insufficiency certain, and that of dilatation highly probable.

Complications.—Gastroptosis is a common complication; it may be secondary to the dilatation or the cause of it. Tetany is a rare, but serious, complication.

Diagnosis.—*Gastroptosis.*—In uncomplicated gastroptosis the position of the stomach is lower down than normal and more or less vertical, but the area of gastric tympany is not increased nor are there present any signs of motor insufficiency.

Type of Dilatation.—A rapid onset, persistent vomiting, pain, a high degree of motor insufficiency with marked dilatation, a tardy inflow and a rapid outflow of water during lavage, active gastric peristalsis, a palpable tumor at the pylorus, and rapid emaciation suggest pyloric obstruction.

Prognosis.—This depends upon the cause, degree, and duration of the dilatation. In pyloric obstruction the prognosis is very grave, although operation may lead to cure, especially in benign forms. Even in atonic dilatation the pronosis must be guarded if the process is advanced and there is pronounced motor insufficiency.

Treatment.—The food should be nutritious, small in bulk, and readily digestible, and, in advanced cases, should be given in small amounts at frequent intervals. Liquids should never be given in large quantities. In severe grades of dilatation, to prevent the tissues from losing water, it is advisable to introduce fluids in the form of water and meat-broth by the bowel. To prevent retention, to control fermentation, and to cleanse the stomach, no measure is so useful as methodic lavage. When there is considerable retention, the lavage should be performed daily, preferably in the early morning. A carefully adjusted abdominal bandage nearly always affords comfort and gives mechanical support to the stomach. In cases due to atony exercise in the open air, hydrotherapy, and, unless there be marked gaseous fermentation, abdominal massage are valuable aids, Faradization of the stomach may also be used to promote muscular contraction. In dilatation from muscular relaxation nux vomica is very useful. Such remedies as creosote, salol, and bismuth-beta-naphthol are sometimes of service in checking fermentation, but they are much less efficacious than systematic lavage. Constipation is best treated by simple enemas or by glycerin suppositories.

Surgical Treatment.—In the large majority of cases of non-obstructive dilatation medical treatment suffices. Occasionally, however, surgical intervention is demanded on account of persistent suffering and progressive emaciation. The operation indicated in these cases is gastro-enterostomy. In cases of pyloric obstruction of a benign character an operation is indicated when it is impossible to maintain nutrition by proper medical treatment. Two operations are available: pyloroplasty and gastro-enterostomy. The treatment of pyloric cancer is considered on page 61.

Acute dilatation of the stomach is the result of paresis of the gastric muscle, the primary cause of which may be traumatism, an abdominal operation, or some debilitating disease, notably pneumonia or typhoid fever. The diagnostic features are acute epigastric distress, abdominal distention, persistent vomiting of large quantities of bilious fluid, a dull area on percussion in the left side of the abdomen, great thirst, and progressive collapse. Unless treatment is soon instituted death usually occurs within a few hours or days. The knee-and-elbow posture, thorough lavage, saline enemas, and appropriate stimulation are the measures most likely to afford relief. The only other resource is operation, which, however, is rarely successful.

GASTROPTOSIS AND ENTEROPTOSIS

(Glénard's Disease)

Definition.—Prolapse of the stomach and transverse colon caused by congenital or acquired weakness of the abdominal muscles and ligaments.

Etiology.—The condition is much more common in women than in men. Tight lacing, repeated pregnancies, abnormal formation of the thorax, enlargement of other abdominal organs, gastrectasis, and constitutional weakness are important predisposing factors.

Symptoms.—The chief objective feature is a more or less pronounced downward displacement of the pylorus, in consequence of which the stomach assumes a vertical or subvertical position. Dislocation of the whole stomach downward is rare. Dilatation of the pyloric extremity is a common sequel. The position and size of the viscus can be determined accurately only by percussion after artificial inflation, or an *x*-ray photograph after the administration of a bismuth mixture. The transverse colon shares in the downward displacement of the stomach and occupies a position immediately below the greater curvature. Ptosis of other abdominal organs, especially of the right kidney and liver, is also present in many cases. Separation of the recti muscles is often seen. A floating tenth rib is less frequent.

The subjective symptoms are those of motor insufficiency or atony of the stomach—fulness and distress after meals, splashing, gaseous eructation, pain in the back, and constipation—combined, in many cases, with more or less marked neurasthenia.

Treatment.—The diet should be adapted to the digestive and motor powers of the stomach. Mechanical support of the pylorus by means of a snugly fitting abdominal bandage affords relief in mild cases. In severe cases, especially if the nervous phenomena are pronounced, a modified rest-cure may prove effective. Lavage is not indicated unless there is general dilatation with retention or excessive secretion of mucus. In very obstinate cases surgical intervention (gastropexy or gastro-enterostomy) should be considered.

HEMATEMESIS

(**Gastrorrhagia**)

Etiology.—Hemorrhage from the stomach may result from (1) traumatism; (2) gastric ulcer or erosion; (3) gastric cancer; (4) venous engorgement of the stomach following cirrhosis of the liver, or splenic enlargement; (5) acute gastritis; (6) atheroma or embolic or thrombotic obstruction of the gastric vessels; (7) blood dyscrasia, as in scurvy, purpura, severe infections, and grave anemias; (8) rupture of an aneurysm; (9) swallowing of blood from the nose, mouth, or throat; (10) vicarious menstruation; (11) hysteria.

Symptoms.—The quantity of blood that is vomited varies considerably: rarely a quart or more is lost. In many cases a portion of the blood escapes through the bowel. The blood is usually dark, is often mixed with food, has an acid reaction, and may be fluid or clotted. If the hemorrhage is severe, the symptoms of acute anemia develop—pallor, weakness, vertigo, tinnitus aurium, dimness of sight, syncope, and convulsions.

Diagnosis.—*Hemoptysis.*—The blood is coughed up; it is usually bright red, frothy, and alkaline in reaction; subsequent expectorations are tinged with blood, and the associated symptoms and signs point to pulmonary or cardiac disease.

Prognosis.—Hematemesis is rarely so severe as to cause death. The most dangerous hemorrhages are those that occur in cirrhosis of the liver, splenomegaly, and aneurysm.

Treatment.—In the treatment of hematemesis absolute rest is essential. No food of any kind should be given by the mouth. An ice-bag should be applied over the stomach, and morphin should be given hypodermically. The application of firm bandages to the four extremities may act favorably. Ergot and such drugs as tannic acid, iron sulphate, and lead acetate are of very doubtful utility. A solution of adrenalin chlorid (1 : 1000) is worthy of trial. Twenty minims may be given in half an ounce of water every hour. When the bleeding is prolonged, 3 or 4 ounces of a solution of gelatin (10 to 15 per cent.) may be given several times a day.

Collapse following hemorrhage will call for diffusible stimulants, the external application of heat, and the subcutaneous or intravenous injection of warm saline solution.

HABITUAL CONSTIPATION

Definition.—Infrequent or difficult evacuation of the feces.

Etiology.—The chief causes are: (1) Sedentary life, lack of exercise, and persistent neglect of the desire for evacuation. (2) Dietetic errors (food deficient in residue, insufficient water, etc.). (3) Many general diseases that weaken the abdominal muscles, lessen the intestinal secretions or inhibit peristalsis, such as anemia, diabetes, neurasthenia, hysteria, melancholia and organic diseases of the brain and spinal cord. (4) Many chronic diseases of the digestive tract—gastric disorders, enteroptosis, chronic intestinal catarrh and obstructive jaundice. (5) Atony of the intestinal musculature from senility. (6) Mechanical obstruction to the passage of the intestinal contents by abnormal kinks (the result of evolutionary or inflammatory bands), stricture, tumors, torsion, etc. (7) Interference with peristalsis by reflex stimuli produced by various painful conditions of the abdominal or pelvic viscera such as irritable prostate, uterine disease, rectal ulcer, anal fissure, hemorrhoids, etc.

The immediate cause of constipation is usually reduced motility in the lower bowel, but in some instances, as in certain cases of hysteria or neurasthenia or of painful pelvic disease it may be spasm of the lower bowel (spastic constipation).

Symptoms.—Some persons continue to enjoy excellent health even though their bowels are evacuated at very infrequent intervals. Usually, however, retention of fecal matter in the intestines longer than is customary with the individual gives rise to unpleasant symptoms, common among which are headache, dizziness, mental sluggishness, lassitude, fetor of the breath, a coated tongue, and anorexia. The diagnosis usually offers no difficulty. Mechanical hindrances are often demonstrable by the *x*-ray.

Sequels.—Severe persistent constipation may lead to piles, fissure, ulceration of the colon, distention of the colon, diarrhea from irritation, fecal impaction, hernia. Straining in constipation may be the immediate cause of apoplexy, attacks of angina pectoris, or the rupture of an aneurysm.

Treatment.—The removal of the cause is a matter of the first importance. Cathartics should be avoided, if possible. In some cases the activity of the bowels is restored by repeated daily attempts at defecation at some special hour. Systematic exercise and cold bathing are of the greatest benefit. Abdominal massage, especially digital kneading in the direction of the colon, is often quite effectual.

Unless the state of digestion offers a contraindication, such laxative articles of food as green vegetables, oatmeal, cornmeal, whole-wheat bread, oils, and cooked fruits should be ordered. Water-drinking should be encouraged. In mild cases a glass of cold water before breakfast may suffice. In constipation arising from a highly refined diet agar (2–6 teaspoonfuls with gruel or cooked fruit) is useful.

General tonics, like iron and strychnin, are sometimes needed. Mineral waters, like Friedrichshall, Hunyadi János, or the milder Saratoga or Bedford waters, are very useful, but possess no special advantages over the saline laxatives (sodium phosphate or Rochelle salt), if the latter are taken in small amounts well diluted. Enemas of soapy water or of glycerin or suppositories of gluten, soap, or glycerin, often prove highly satisfactory. In obstinate atony systematic colonic irrigation with cold water is sometimes efficacious. In cases of fecal impaction enemas of warm oil (4–6 ounces, at night) will be found useful. Vegetable cathartics are usually necessary in obstinate cases. The mild ones should always be tried first, and even with these considerable care should be exercised lest the patient comes to rely upon drugs to the exclusion of the hygienic and dietetic measures already indicated. Of the mild laxatives, cascara sagrada is one of the best: from 10 to 30 minims of the fluid extract, or a corresponding dose of a pleasant elixir, may be administered at bedtime and repeated, if necessary, in the morning. In constipation resulting from constrictions of a spastic or an organic nature, liquid paraffin (1–2 tablespoonfuls morning and night, on an empty stomach, the dose being gradually reduced) often affords relief.

In many cases a combination of several laxatives (rhubarb, aloes, podophyllum, euonymin, and colocynth) acts better than any one singly. As adjuvants, nux vomica or physostigma may be added to overcome intestinal atony, and belladonna or hyoscyamus to prevent griping. The most suitable combination must be determined in each case by experience. A pill, like one of the following, will generally prove satisfactory:

℞.	Aloini	gr. iv
	Strychninæ sulphatis	gr. ⅕
	Extracti belladonnæ	gr. iij.—M.

Fiant pilulæ No. xxiv.
Sig.—One pill at bedtime.

℞.	Pulveris rhei	
	Extracti rhamni purshianæ	āā gr. xxiv
	Extracti euonymi	gr. xij
	Extracti physostigmatis	
	Extracti belladonnæ	āā gr. iv.—M.

Fiant pilulæ No. xxiv.
Sig.—One pill at bedtime.

In extreme cases ileo-sigmoidostomy, with or without excision of the colon, should be considered.

INTESTINAL COLIC

(Enteralgia; Tormina)

Definition.—Intestinal pain of a spasmodic character.

Etiology.—It usually results from irritating food, flatulence, or fecal accumulation. It is a common symptom of structural lesions of the bowel—enteritis, dysentery, appendicitis, intestinal obstruction. It is an important symptom in chronic lead-poisoning. It may be reflex from disease of the ovaries, uterus, liver, vertebræ, etc. It may occur as a crisis of locomotor ataxia.

Symptoms.—The chief feature is paroxysmal pain of a cramp-like character, centering around the umbilicus, and relieved by pressure. The abdomen is usually distended. Severe attacks may lead to collapse, indicated by cold sweats, pinched features, feeble pulse, and vomiting. The attack lasts from a few minutes to several hours, and usually ends with a discharge of flatus.

Diagnosis.—*Lead Colic.*—This may be recognized by the history, blue line on the gums, retracted abdominal wall, wrist-drop, and lead in the urine.

Biliary Colic.—Pain radiating from the liver to the back, jaundice, enlargement and tenderness of the gall-bladder, and calculus in the stool are characteristic of gall-stone colic.

Renal Colic.—This is indicated by pain extending from the kidney, along the ureter to the penis and testicle, frequent micturition, blood or calculus in the urine.

Chronic Appendicitis.—Localized tenderness (McBurney's point), muscular rigidity, and induration point to appendicitis.

Intestinal Obstruction.—In this condition vomiting is incessant and often of a stercoraceous character; constipation is absolute, without discharge of feces or flatus, tumor is sometimes present, and collapse symptoms are marked.

Acute peritonitis is revealed by continuous pain, fever, abdominal tenderness, rigidity, and leukocytosis.

Treatment.—The indications are to relieve pain and to remove the cause. Turpentine stupes are useful. In severe cases it will be necessary to give morphin (1/8 to 1/4 grain) and atropin (1/100 grain) hypodermically. Carminatives—peppermint, ginger, oil of cloves, Hoffman's anodyne—often afford relief.

Colic excited by irritating food or fecal accumulation is promptly relieved by saline or mercurial purges.

DIARRHEA

Definition.—A condition in which the stools are too frequent and too liquid. Like dyspepsia, it is a symptom of many pathologic conditions.

Etiology.—(1) It is sometimes due to excessive ingestion of water or of certain foods (fats, fruits, coarse vegetables). (2) It is sometimes produced by diseases of the stomach, as incontinence of the pylorus and achylia gastrica. (3) It results from inflammation of the intestines—enteritis, ileocolitis, dysentery (inflammatory diarrhea). (4) It is a symptom of certain infectious diseases, such as typhoid fever and cholera (symptomatic diarrhea). (5) It may be excited by cathartic drugs. (6) It often occurs as a final symptom in cachectic states, as in cancer, diabetes, and chronic Bright's disease (colliquative diarrhea). (7) It sometimes marks the crisis of acute infections, such as typhus fever and pneumonia (critical diarrhea). (8) It may result from certain nervous influences (emotional excitement, Graves' disease, neurasthenia).

ACUTE ENTERITIS

Etiology.—The occurrence of acute catarrhal inflammation of the intestinal mucous membrane is favored by hot weather, bad hygienic surroundings, and improper food. Chilling of the surface may also be a factor. The direct cause is usually some irritant produced in food through the action of bacteria. Less frequently bacteria themselves are the exciting factors. The *Bacillus coli communis*, *Bacillus enteritidis*, paracolon bacillus, *Bacillus dysenteriæ* (Shiga) or allied organisms, and, less frequently, the *Bacillus botulinus*, streptococci or staphylococci, may be the active agents. Acute enteritis may also be excited by mineral poisons (antimony, arsenic, mercury) and by certain autogenic poisons (uremia). As a secondary process it is observed in many infections, as typhoid fever, cholera, dysentery, etc.

Pathology.—The mucous membrane is swollen, injected, and covered with mucus. The lymph follicles are enlarged and not rarely show erosion or actual ulceration. In severe cases small hemorrhages occur and exceptionally the mucous membrane is covered with a more or less extensive false membrane.

Symptoms.—The stools are frequent—three or four to twenty or more a day—watery, brownish, and, as a rule, more or less offensive. The amount of visible mucus is rarely considerable unless the large bowel is especially affected. Undigested food may be present if the upper part of the small bowel is chiefly involved. Accompanying the diarrhea there are usually colicky pains, abdominal soreness, tympanites, borborygmus, and more or less fever, although the temperature may be normal. Nausea and vomiting may be marked if the stomach is also affected, and sometimes jaundice develops from the extension of the inflammation from the duodenum into the bile-ducts. Tenesmus is absent unless the inflammation extends to the lower colon.

Cholera Morbus (Cholera Nostras).—This is a very severe form of acute enteritis usually traceable to food poisoning. It is characterized by persistent bilious vomiting, frequent and copious watery stools, violent abdominal pains of a cramp-like character, marked pyrexia, great prostration and, in extreme cases, collapse. Death rarely occurs, however, except in aged or debilitated subjects.

Diarrhea in Childhood.—The predisposing factors comprise warm weather, improper food, bad hygienic conditions, dentition and malnutrition. Qualitative changes in the food, especially in milk, the result of bacterial action, is the most common exciting cause. Three types are usually recognized: (1) Simple or dyspeptic diarrhea; (2) ileocolitis; (3) cholera infantum. The line between these types, however, cannot be sharply drawn.

Simple or Dyspeptic Diarrhea.—In this form the stools are offensive and usually contain undigested curds of milk, but little or no visible mucus. Vomiting frequently occurs. The temperature generally ranges from 100° to 103° F. Unless the disease is long continued prostration and emaciation are not marked. Recovery is the rule, although relapses are common and the condition may lead to ileocolitis.

Ileocolitis.—In this form of diarrhea the symptoms are more intense. The stools are numerous and contain much mucus and not rarely streaks of blood. The abdomen is distended and tender. The temperature usually ranges from 103° to 105° F. Vomiting often occurs, but usually it is a less conspicuous feature, than in the dyspeptic form. Colicky pains may precede the stools and not infrequently there is pronounced tenesmus. The *Bacillus dysenteriæ* is found in some cases. Exhaustion and emaciation rapidly ensue, and in severe cases the child sinks into a state of complete collapse or passes into a marantic state persisting several days. Death is frequently preceded by extreme apathy, stupor or even coma (spurious hydrocephalus). Bronchopneumonia and acute nephritis are not uncommon complications. The disease is always a serious one, especially in young infants, and even in favorable cases convalescence is likely to be tedious.

Cholera Infantum.—This is the rarest but the gravest form of summer diarrhea. The onset is usually sudden. Vomiting and purging begin almost simultaneously and become incessant. The stools are large and watery. Thirst is intense. The surface temperature is low but the rectal temperature is very high (105°–106° F.). The urine is scanty or suppressed. Collapse soon follows, and is shown by pinched features, hollow eyes, sunken fontanel, pallid skin and cold surface. Even at this time a reaction may set in, but in the large majority of cases death results in from twenty-four to forty-eight hours. Toward the close stupor, convulsions, and coma (spurious hydrocephalus) may supervene.

Diagnosis.—The diagnosis of acute enteritis usually offers no great difficulty. The gradual onset, nose-bleed, splenic enlargement, continued fever, general prostration, and serum reaction will lead to the recognition of *typhoid fever*. *Peritonitis* may be distinguished by the more intense pain and tenderness, greater tympanites, constipation, and marked constitutional disturbance. As to the location of the inflammation, especial *involvement of the small bowel* is suggested by vomiting, borborygmus, and the occurrence of large fecal stools containing particles of undigested food but little or no visible mucus; *colitis* is suggested by tenderness along the course of the colon, tenesmus, and the occurrence of blood or of large amounts of mucus in the evacuations.

Treatment.—Rest in bed and the substitution of bland nourishment for the ordinary diet are all that is required in many cases. Boiled milk, milk and arrow-root, and mutton, veal, or chicken broth are suitable foods. In robust subjects it may be advisable to withhold all food for twenty-four or thirty-six hours. If the patient is seen at the outset and there is reason to believe that irritant material is still present in the bowel, an unirritating purgative, such as castor oil, Epsom salts, or calomel should be given. Occasionally a second dose of the purgative may be given with benefit. Externally, sinapisms or stupes are frequently efficacious. If the diarrhea continues mild astringents (bismuth subnitrate or chalk) and opium are indicated. They may often be combined advantageously with antiseptics as in the following formulas:

℞.	Morphinæ sulphatis		gr. j
	Phenylis salicylatis		gr. xxiv
	Bismuthi subnitratis		℥ss.—M.

Fiant chartulæ No. xii.
Sɪɢ.—One every three hours.

℞.	Bismuthi subsalicylatis		ʒiss
	Cretæ præparatæ		ʒijss
	Tincturæ opii camphoratæ		f℥i
	Pulveris acaciæ	q. s.	
	Aquæ cinnamomi	q. s. ad	f℥vi.—M.

Sɪɢ.—A tablespoonful every three hours.

In *cholera morbus* the hypodermic injection of morphin (1/4 grain) and atropin (1/100 grain) is necessary. Hot applications to the abdomen are useful. Calomel in fractional doses serves to allay vomiting and to rid the bowel of irritating matter. Collapse will require hot bathing, diffusible stimulants (ammonia, brandy, camphorated oil), and in some instances subcutaneous injections of salt solution.

Acute Enteritis in Infants.—The first indication is to withdraw the milk at once, and to withhold it for several days or until the stools become quite natural. Indeed, in many cases it is well to suspend all nourishment for the first twenty-four hours, allowing nothing by the mouth but barley-water or plain boiled water. Subsequently, albumin-water, fresh beef-juice, veal broth, or a liquid peptone preparation may be given in lieu of milk. Milk feeding should always be resumed very gradually. Absolute rest in the recumbent position is essential. Removal to the seashore or mountains is often of the greatest benefit.

To remove irritant matter from the bowel, castor oil or calomel should be given, preferably the latter when the stomach is sensitive. In most cases it is necessary to follow the purge with a sedative astringent like bismuth subnitrate or chalk. From 5 to 10 grains of one of these drugs may be given every two or three hours with an intestinal antiseptic (salol, beta-naphthol-bismuth, bismuth salicylate); some such combination as the following may be ordered:

℞.	Bismuthi subnitratis	ʒij–iv
	Salol	gr. xxiv
	Misturæ cretæ	f℥iij.—M.

Sig.—A teaspoonful every two hours.

A more active astringent, like tannalbin or tannigen (2 to 3 grains), may be given in addition to the bismuth subnitrate or chalk when the discharges are exceedingly profuse and watery.

Opium is often of great value, but extreme caution must be exercised in its use. It is called for when the diarrhea continues in spite of the thorough unloading of the bowel and the administration of mild astringents. From 3 to 5 minims of paregoric may be given every two, three, or four hours, according to circumstances. If the stomach is unretentive, laudanum (1 to 2 minims) may be given by enema.

In *ileocolitis* intestinal irrigation is an important part of the treatment. Once or twice a day the colon should be thoroughly flushed with saline solution or with water containing a dram ful cases, a digital examination supplemented, if necessary, by proctoscopy, should be made in order to exclude carcinoma of the rectum.

Prognosis.—The prognosis is uncertain. If the cause can be removed, however, a complete cure may be effected.

Treatment.—The cause must be ascertained and removed, if possible. The diet, clothing, habits, occupation, and mode of living of the patient should receive careful attention. No definite rules can be laid down in reference to the diet. When the disease is not very severe and is confined for the most part to the colon, a selected mixed diet may be allowed. Many patients do well upon an exclusive milk diet. Foods that are bulky and leave much residue are always inadmissible.

Protection of the body against chilling is of vital importance. Woolens should be worn next to the skin. A snugly fitting abdominal bandage may be worn as an additional safeguard. Rest in bed is sometimes essential. When the general nutrition is not too much impaired, a change of air and scene may prove very beneficial.

Mineral astringents, especially bismuth subnitrate (30 to 40 grains), silver nitrate (¼ to ½ grain), copper sulphate (¼ to 1 grain), and lead acetate (1 to 3 grains) are of service.

Intestinal antiseptics—salol, bismuth salicylate, beta-naphthol-bismuth—are useful adjuvants. Opium is often required in acute exacerbations. When the disease is situated chiefly in the colon, irrigation of the bowel two or three times a week with a solution of silver nitrate (10 to 20 grains to 1 pint) is especially to be recommended.

MUCOUS COLITIS

Definition.—Mucous colitis, or mucous colic, is a chronic secretory neurosis of the colon, characterized by the regular or periodic passage of masses of mucus and paroxysms of abdominal pain.

Etiology.—The disease is most common in middle life, and affects women much more frequently than men. It is met with particularly in nervous, neurasthenic, or hysterical individuals, who suffer also from indigestion and constipation. In some cases it is secondary to organic disease of the bowel, as chronic appendicitis, tumor, or stricture.

Symptoms.—The chief phenomena are constipation, the passage of excessive quantities of mucus in the form of flakes, shreds or casts of the bowel, indigestion, and paroxysmal attacks of intestinal colic. Symptoms of hypochondriasis, neurasthenia, or hysteria are also present in greater or less degree in most cases. Emaciation is sometimes marked.

Prognosis.—The disease is very obstinate, but rarely fatal. Marked improvement or even recovery may follow treatment.

Treatment.—The diet should be abundant and varied. Bulky foods are recommended. The general treatment laid down for neurasthenia or hysteria is usually applicable. Mild laxatives, preferably frequent doses of castor oil, should be given. Tincture of nux vomica in ascending doses with sodium bicarbonate is sometimes of service. Mild intestinal antiseptics may be beneficial. Colonic douches, with warm alkaline water or solution of silver nitrate (1 : 6000), or high injections of cotton-seed oil (6 ounces), are often helpful.

SPRUE

Sprue, or psilosis, is a chronic inflammatory disease of the alimentary canal, occurring in certain tropical countries, and characterized by aphthous stomatitis and glossitis, dyspeptic symptoms, frequent, copious, and yeasty-looking stools, and severe wasting and anemia. The cause is unknown. Recovery usually ensues when the patient is kept at rest and restricted to a milk diet, but a tendency to relapse is very pronounced.

APPENDICITIS

(Typhlitis; Perityphlitis)

Definition.—An inflammation of the appendix vermiformis.

Pathology.—There are three varieties: Catarrhal, ulcerative, and interstitial.

Catarrhal Appendicitis.—In mild cases the appearances are, no doubt, similar to those observed in catarrh elsewhere, but in severe cases the wall of the appendix is infiltrated with round-cells, and the mucous membrane is denuded of epithelium and presents a granular surface. This latter condition may eventuate in diffuse chronic appendicitis with relapses (*recurrent appendicitis*), or union of the granulating surfaces with complete obliteration (*appendicitis obliterans*).

Ulcerative Appendicitis.—In this type the wall of the appendix is the seat of a more or less localized ulcer. It may be associated with the presence of fecal concretion or a foreign body, or it may be the result of typhoid or tubercular infection.

Interstitial Appendicitis.—In this form mucous, submucous, and muscular coats are swollen, infiltrated, and the seat of necrotic changes, and the peritoneal covering is congested and roughened. Suppuration may ensue, transforming the appendix into a pus-containing sac, or the walls of the organ may become gangrenous. In either case spontaneous rupture is likely to occur. In other instances a chronic process follows, in which the appendix becomes thickened, indurated, and adherent to adjacent structures.

Appendicitis is always due to the action of pathogenic bacteria, the chief offenders being the Bacillus coli communis, Streptococcus pyogenes, Staphylococcus pyogenes aureus, typhoid bacillus, and tubercle bacillus. Of these, the Bacillus coli communis, a natural habitant of the bowel, is most commonly present. Under ordinary conditions it is harmless, but if the circulation of the appendix is interfered with from any cause or the coats of the tube are abraded, infection is liable to arise.

Etiology.—Appendicitis is more common in males than in females. It is most frequent between the fifteenth and thirtieth years. Exposure, errors in diet, intestinal catarrh, traumatism, and the lodgement in the appendix of fecal concretions or foreign bodies predispose to the disease. It may follow some infection like typhoid fever, influenza, or tuberculosis. It may be induced by twisting of the appendix.

Symptoms.—These comprise: (1) Sudden pain, often general at first, but later most marked in the right iliac region. (2) Circumscribed tenderness, most frequently detected over McBurney's point—a point midway on a line between the umbilicus and the anterior superior iliac spine. (3) Fever, ranging between 100° and 103° F. (4) Localized rigidity in the right iliac fossa, or the presence of a definite tumor. (5) Reflex disturbances—nausea, vomiting, and constipation, or, rarely, diarrhea. (6) Leukocytosis of from 12,000 to 20,000 per c.mm.

Terminations.—Resolution, general peritonitis, and localized abscess. The location of the abscess depends on the position of the appendix. It may be found in either of the lower quadrants or beneath the diaphragm (subphrenic abscess). The pus may be discharged through the abdominal walls, the bowel, bladder, or vagina, or it may escape into the tissues of the lumbar region or thigh. Appendicitis occasionally excites hepatic abscess, the infection being carried through the portal vein.

Diagnosis.—*Typhoid Fever.*—The gradual onset, characteristic temperature-curve, epistaxis, mental hebetude, diarrhea, splenic enlargement, absence of leukocytosis, and, later, the rash and Widal reaction will indicate typhoid fever.

Renal Colic.—This may be recognized by the absence of fever and of local rigidity, and the presence of hematuria.

Acute Inflamation of the Gall-Bladder.—Pain and tenderness in the right hypochondrium, a smooth, mobile tumor, and a history of biliary colic would suggest this condition.

Tubal Disease.—The history and results of pelvic examination will usually prevent an error in diagnosis.

Prognosis.—The prognosis depends upon the type. The average mortality is about 14 per cent.

Treatment.—The patient should be kept in bed at absolute rest. All food should be withheld until the acute symptoms have subsided. Constipation is best relieved by enemas of warm water. Locally, cold or heat may be applied, according to the sensations of the patient. The use of morphin is generally discredited, since it tends to mask the symptoms.

An operation should be urged—(1) At once in all cases in which the onset is very severe, the symptoms indicating special severity being marked right-sided tenderness and rigidity, distention, and vomiting, with or without fever; (2) in cases of moderate severity which manifest no improvement after the lapse of forty-eight hours; and (3) in cases in which the symptoms, after decided improvement, return. Operation during the quiescent stage, when the element of danger is almost entirely removed, is to be recommended—(1) If an acute attack has been followed by persistent tumefaction and tenderness, intestinal disturbances, or impairment of the general health; (2) if there have already been two attacks, even of moderate severity; and (3) if mild attacks occur with such frequency as to induce disability.

INTESTINAL OBSTRUCTION

(Ileus)

Intestinal obstruction may be either acute or chronic. The chief causes of the *acute form* are: (1) Strangulation; (2) intussusception; (3) volvulus; (4) impaction of foreign bodies or gall-stones; (5) paresis of the intestine; (6) congenital malformation or stricture.

Chronic obstruction may be due to—(1)Impaction of feces; (2) stricture; (3) tumors of the bowel or of neighboring organs.

Symptoms of Acute Obstruction.—These consist of: (1) Sudden abdominal pain—at first paroxysmal, but later continuous; (2) constipation, soon becoming absolute; (3) vomiting, persistent and ultimately of a stercoraceous character; (4) abdominal distention; (5) visible peristaltic waves; (6) collapse, indicated by pinched features, sunken eyes, a cold, mmy surface, and a frequent feeble pulse.

Symptoms of Chronic Obstruction.—The symptoms usually develop gradually. Acute symptoms may appear, however, when the occlusion becomes complete. The chief features are intractable constipation, colicky pains, distention of the abdomen, and gradual failure of health. The stools may be ribbon-shaped or in the form of scybalous masses, and are sometimes coated with mucus and blood. Vomiting is not common.

Diagnosis.—Early vomiting, slight distention, suppression of urine, and rapid collapse point to an obstruction *high in the small intestine.*

Acute Generalized Peritonitis.—The history, early appearance of fever and of diffuse tenderness, signs of effusion, and absence of stercoraceous vomiting will indicate peritonitis.

Strangulation.—This often occurs in external hernia, when it can be recognized by an examination of the inguinal, femoral, and umbilical rings.

Internal strangulation is very common. It may be due to the slipping of a coil of intestine under bands of adhesions, the result of antecedent peritonitis, or under Meckel's diverticulum that is abnormally attached to the abdominal wall, or through a slit in the omentum or mesentery, the foramen of Winslow, or the diaphragm. It usually occurs in young adults; there is often a history of injury or of peritonitis, and the symptoms are very acute.

Intussusception or Invagination.—This is the slipping of a portion of the intestine into the part immediately below it. It occurs especially in children. Its exciting cause is probably irregular peristalsis, whereby one part of the bowel is constricted while the adjoining part is dilated. The usual seat is the ileocecal region.

Multiple invaginations are frequently found postmortem, which have resulted from the irregular peristalsis occurring just before death; they possess no inflammatory characteristics. In invaginations not cadaveric the parts are injected, swollen, and covered with lymph.

DISEASES OF THE PANCREAS

ACUTE PANCREATITIS

Varieties.—Hemorrhagic, suppurative, and gangrenous.

Etiology.—Acute pancreatitis may result (1) from cholelithiasis, due to the extension of an infectious inflammation from the biliary tract into the pancreatic duct or to the entrance of bile into the pancreas, as when a gall-stone is so lodged in the diverticulum of Vater that the common bile-duct and the pancreatic duct become a continuous closed channel; (2) from inflammatory affections in adjacent parts—gastro-duodenal catarrh, gastric ulcer, or cancer; (3) from general infections—specific fevers and pyemia; (4) from traumatism. Many of the patients are fat and have used alcohol in excess.

Pathology.—In the hemorrhagic form the organ is irregularly enlarged and the seat of hemorrhagic extravasation. Opaque, white spots of a tallowy consistence are frequently found in the interlobular tissue, omentum, and surrounding parts, and represent areas of *fat necrosis.*

Suppurative pancreatitis may occur as a primary condition or as a sequel of the hemorrhagic form. There may be multiple abscesses or one large collection of pus. More or less extensive areas of necrosis are found. Thrombosis of the portal or splenic veins is frequently encountered. Pancreatic abscesses may become encapsulated or they may rupture into the peritoneum, stomach, or duodenum.

Gangrenous pancreatitis is usually secondary to one of the other varieties.

Symptoms.—Hemorrhagic pancreatitis is marked by sudden intense pain in the upper part of the abdomen often radiating to the back; distention of the epigastrium, with localized tenderness and rigidity; vomiting of bile-stained mucus, or occasionally, of bloody material, and symptoms of profound collapse. Constipation is the rule, but diarrhea is not uncommon. Slight jaundice is often observed. Fatty stools and glycosuria are very rarely present. Death usually occurs in from one to three days, but sometimes the severity of the symptoms diminishes and the disease enters upon a stage of necrosis (gangrene) or of suppuration extending over several weeks or months. This transition is indicated by a tumor mass in the epigastrium, irregular fever, leukocytosis, and progressive weakness and emaciation. Jaundice and chills may also occur.

Occasionally in primary suppurative pancreatitis the onset is gradual, and for many months the only symptoms are abdominal pain and digestive disturbances. In other cases, however, jaundice, fever, chills, diarrhea, emaciation, and a tumor mass in the epigastrium are also present.

Diagnosis.—*Intestinal Obstruction.*—In this condition the onset is usually less severe, fecal vomiting is common, pain and distention are less frequently limited to the epigastrium, and constipation is absolute, not even flatus being passed.

In *perforation of a gastric or duodenal ulcer* the antecedent history usually presents some of the characteristic features of ulcer, and although the patient's face is pallid and anxious, his pulse, at least in the first two or three hours, is very much less altered either in frequency or volume than in acute pancreatitis. In *biliary colic* the symptoms of collapse are less pronounced, the pain, tenderness, and rigidity are chiefly in the region of the gall-bladder, and the extreme restlessness of the patient are in striking contrast with the motionless rigidity observed in acute pancreatitis. In *ruptured extrauterine pregnancy* the history, the results of vaginal examination, and the rapidly increasing pallor and faintness with sighing inspiration, restlessness, etc., will usually determine the diagnosis.

Prognosis.—This is very unfavorable. The duration varies from a day or two in the hemorrhagic form, to several weeks in the chronic suppurative variety. Recovery may follow operation or rupture of the abscess into the bowel. The disease rarely ends in chronic pancreatitis.

Treatment.—Operation after the initial collapse offers some hope of cure.

CHRONIC PANCREATITIS

(Cirrhosis of the Pancreas)

Etiology.—It may result—(1) From closure of the pancreatic duct by gall-stones impacted in the common bile-duct; (2) from extension of inflammation in gastro-duodenal catarrh or pyloric ulcer; (3) from syphilis or alcoholism; (4) from sclerosis of the pancreatic arteries, and, possibly, (5) from acute pancreatitis.

Pathology.—The chief lesions are an overgrowth of the fibrous tissue and more or less degeneration or atrophy of the cellular elements.

Symptoms.—The symptoms are obscure. Flatulent dyspepsia, paroxysmal epigastric pain, a tendency to diarrhea, slight jaundice, and progressive wasting are the usual features. Fatty stools have been noted in a few instances. When the cirrhotic process is diffuse (chronic interacinous pancreatitis) and implicates the islands of Langerhans, the symptoms of *diabetes mellitus* develop, otherwise glycosuria is rare.

Prognosis.—The disease runs a slow course. If glycosuria develops, the outlook is more grave.

Treatment.—The use of fats and starches should be restricted. Carbonated waters are said to increase pancreatic secretion. Pancreatin is recommended. Surgical treatment offers a good chance of recovery in gall-stone cases.

CANCER OF THE PANCREAS

Etiology.—The disease most frequently occurs in males past forty years of age.

Pathology.—Pancreatic cancer generally involves the head of the gland, and is commonly of the scirrhous variety. It may be primary or secondary.

Symptoms.—These include disturbances of digestion, rapid loss of flesh and strength, anemia, severe deep-seated epigastric pain, and the presence of a tumor. The latter is usually found a little above the navel; it is but slightly movable, deep seated, and often pulsatile from its relation to the aorta. The pain often occurs in paroxysms, especially at night, and may be associated with the symptoms of collapse. Progressively increasing jaundice, with enlargement of the gall-bladder, is a frequent symptom, and results from the pressure of the tumor upon the common bile-duct. Pressure on the portal vein may cause ascites. Glycosuria is an occasional symptom. In some cases the stools have contained much free fat and numerous undigested muscle-fibers.

Diagnosis.—*Gastric Cancer.*—In this condition the tumor is more freely movable, is usually associated with dilatation of the stomach and with marked gastric symptoms. Pain is not usually so severe. Jaundice is rare.

Gall-stone in Common Duct.—In this condition jaundice is likely to be remittent, febrile paroxysms are often present, there is no epigastric tumor, the gall-bladder is usually small, and emaciation is less rapid.

CYSTS OF THE PANCREAS

Varieties.—(1) Retention cysts from impaction of a calculus, stricture, or tumor; (2) traumatic cysts from hemorrhagic extravasation; (3) proliferation cysts (carcinomatous or adenomatous).

Pathology.—Pancreatic cysts, as a rule, grow forward and upward, and project between the stomach and colon. The contents, which are commonly viscid, may be clear or bloody. Pancreatic ferments are often present.

Symptoms.—These are very variable, the most common being deep-seated epigastric pain, digestive disturbances, vomiting, and emaciation. Jaundice sometimes occurs from compression of the bile-duct, and less frequently ascites from compression of the portal vein. Free fat and undigested muscular fiber may be found in the stools and sugar in the urine. Physical examination often reveals in the upper part of the abdomen, between the stomach and the colon, a smooth, rounded, fluctuating tumor.

Prognosis and Treatment.—The prognosis is guardedly favorable under operative treatment.

PANCREATIC CALCULI

Pancreatic calculi are probably due to infection of the pancreatic ducts and stagnation of the pancreatic secretion. Their passage through the duct excites *pancreatic colic*, the symptoms of which resemble biliary colic, but the pain is more likely to radiate to the left and is usually unattended with jaundice. The coexistence of glycosuria, with fatty stools, and the discovery in the stools of concretions containing chiefly carbonate or phosphate of lime, would confirm the diagnosis.

DISEASES OF THE LIVER

Area of Liver Dulness.—The absolute dulness (part uncovered by lung) extends in the mammary line from the upper border of the sixth rib to the costal margin; in the axillary line, from the seventh rib to the eleventh rib; in the scapular line, from the ninth rib to the eleventh rib; in the median line, the upper border is lost in the cardiac dulness, while the lower border lies midway between the ensiform cartilage and the umbilicus. Slight dulness in the mammary line begins at the fifth rib.

Palpation.—*Palpation* of the liver is practised to determine position, size, form, and consistence and to detect any tenderness or pulsation.

Conditions in which the liver is palpable:

1. In thin subjects the edge is sometimes palpable under normal conditions.

2. In very young children in whom the liver is always proportionately large.

3. In depression of the liver, as by a pleural effusion or by a consolidated lung.

4. When the suspensory ligaments are relaxed and the liver "wanders."

5. In enlargement of the organ from any cause.

6. In certain abnormalities of form, as in the "corset liver."

Superficial Irregularities.—Small irregularities may be noted in cancer of the liver, syphilis of the liver, and very rarely in atrophic cirrhosis.

Large prominences are sometimes noted in tumors, abscesses, and hydatid cysts.

Consistence.—The liver is firm to the touch in hypertrophic cirrhosis, cancer, congestion, leukemic infiltration, and amyloid disease. In abscess and hydatid disease the resistance is less marked and sometimes fluctuation can be noted.

Tenderness.—The liver is tender in congestion, abscess, cancer, hypertrophic cirrhosis, and in affections complicated with perihepatitis.

Pulsation may be detected in the venous congestion resulting from tricuspid regurgitation, in abdominal aneurysm, and in tumors of the left lobe resting on the aorta.

Percussion.—Percussion determines size and resistance.

The liver is uniformly enlarged in: (1) Congestion, active and passive. (2) Fatty infiltration. (3) Amyloid infiltration. (4) Cirrhosis, especially hypertrophic. (5) Leukemic infiltration. (6) Infiltrating carcinoma. (7) Suppurative cholangitis.

Irregular enlargements of the liver are noted in: (1) Nodular cancer. (2) Abscess. (3) Hydatid disease. (4) Syphilis.

The *liver is diminished in size* in: (1) Atrophic cirrhosis, late stage. (2) Fatty degeneration. (3) Acute yellow atrophy. (4) Senile atrophy. The area of hepatic dulness may be diminished from certain extrinsic causes, namely, pulmonary emphysema, excessive tympanites, and perforation of the stomach or bowel.

JAUNDICE OR ICTERUS

Definition.—Pigmentation of the tissues and excretions with bile-pigments.

Varieties.—(1) Obstructive jaundice. (2) Toxemic jaundice.

Etiology of Obstructive Jaundice.—Obstruction to the outflow of bile leads to its accumulation in the biliary passages and reabsorption into the blood.

Obstruction may be due to the following causes:

1. Stricture of the bile-duct, congenital or acquired.
2. Catarrh of the bile-ducts or of the duodenal mucous membrane around the orifice of the ductus choledochus.
3. Foreign bodies in the ducts, as gall-stones or parasites.

4. Tumors of the liver or of adjacent viscera compressing the ducts.

5. Compression of the ducts by enlarged lymph glands, inflammatory adhesions, or fecal accumulations.

6. Kinking of the ducts the consequence of gastroptosis, nephroptosis, abdominal tumors, the pregnant uterus, etc.

7. Spasm of the bile-ducts. This has been advanced as the cause of the jaundice that rarely follows emotional excitement.

Symptoms.—The skin, mucous membranes, and secretions are stained yellow. The discoloration is usually first noticed in the conjunctivæ. The stools are pale, fetid, and fatty; the urine is dark, and in long-standing cases may contain hyaline casts. The pulse rate is often reduced, and the temperature may be slightly subnormal. There is more or less mental depression, and in chronic cases delirium, convulsions, and coma occasionally develop. Itching of the skin is often noted, and urticaria is a common complication. In grave cases subcutaneous ecchymoses may appear. The clotting time of the blood is much increased.

Diagnosis.—Other discolorations, like the bronze hue of Addison's disease, the sallow complexion of advanced cachexia, the pigmentation of the skin in hemochromatosis and the green tint of chlorosis, may resemble jaundice, but in these cases the conjunctiva remains white and the urine lacks bile.

Etiology of Toxemic Jaundice.—This form of jaundice occurs (1) in certain intoxications as in poisoning by toluylendiamin, phosphorus or snake-venom; it may be observed (2) in various infections, such as yellow fever, relapsing fever, septicopyemia, etc.; it occurs (3) in acute yellow atrophy of the liver and in Weil's disease; (4) it is occasionally seen in certain conditions associated with excessive hemolysis, as pernicious anemia and paroxysmal hemoglobinuria.

In toxemic jaundice there is also obstruction, but it is situated in the minute bile-ducts instead of in the larger ones.

The chief cause of the obstruction is probably catarrhal inflammation (cholangitis), excited by the poison circulating in the blood, although extensive destruction of the red blood-cells, which leads to the secretion of a thick, viscid bile, rich in pigments (polychromia), is doubtless a contributing factor in many cases.

Symptoms.—These are much the same as in obstructive jaundice, but the staining of the skin is not so intense, and the stools still contain bile. Grave constitutional symptoms (delirium, stupor, multiple hemorrhages, etc.) are more frequent, and other evidences of the underlying infection or intoxication are usually present.

ICTERUS NEONATORUM

Physiologic icterus in the new-born is slight. According to Quincke, it is probably due to the passage of a part of the portal blood rich in bile-pigments directly into the vena cava by way of the ductus venosus, which remains patent for several days after birth.

Pathologic icterus in the new-born is marked, and commonly proves fatal. It results from congenital stricture of the bile-ducts, syphilis of the liver, septic infection through the umbilical vein, or epidemic infection (Winckel's disease).

CHOLEMIA

The term cholemia has been used to designate the grave intoxication which sometimes develops in severe forms of jaundice and in the later stages of liver disease even in the absence of jaundice. The condition is characterized by headache, delirium, stupor, coma, convulsions, and not rarely, by hemorrhages into the skin and from the mucous membranes. These symptoms are probably due to the retention in the blood of poisonous compounds which are normally rendered non-toxic by the liver, although an underlying or concomitant infection may be a factor in some cases.

ACHOLURIC JAUNDICE

A yellowish discoloration of the skin and conjunctivæ is sometimes seen in cases of pronounced urobilinuria in which the urine is free from bile. This discoloration is not due to urobilin pigmentation, however, but is a true jaundice (*acholuric jaundice*), bile pigment being invariably present in the blood but in quantities too small to be excreted in the urine. Jaundice of this type is the most conspicuous symptom in *chronic family jaundice*, a comparatively rare disease, which tends to affect several members of a family and is usually congenital, although it may attack but one of a family and may appear first in adolescence. The disease is characterized by persistent jaundice, enlargement of the spleen, moderate anemia, and attacks of acute indigestion or of abdominal pain, due in some instances to complication with gall-stones. The stools are well colored and the urine contains urobilin but no bile. The liver is not at all or only slightly enlarged and signs of constitutional disturbances are generally lacking. The symptoms are probably due to increased fragility of the erythrocytes. Other conditions that must be ruled out in making the diagnosis are congenital stenosis of the bile-ducts, the infectious forms of icterus, syphilitic hepatitis and splenic anemia. So far as life is concerned the prognosis is good. In a number of instances splenectomy has effected a cure.

CATARRHAL CHOLANGITIS

(Catarrhal Jaundice; Catarrh of the Bile-ducts)

Etiology.—(1) The disease is usually met with in young adults as a sequel of a gastro-intestinal catarrh, which has been excited by dietetic indiscretions or exposure to cold. (2) It is an occasional accompaniment of infectious diseases, as pneumonia and typhoid fever. (3) It is often associated with cholelithiasis or organic disease of the liver. (4) It rarely occurs in epidemic form.

Pathology.—The large ducts are particularly affected; the mucous membrane is swollen and covered with tenacious mucus. When the gall-bladder is compressed, bile is ejected through the duodenal orifice with less ease than is natural.

Symptoms of Acute Catarrhal Cholangitis.—(1) Symptoms of gastro-duodenal catarrh usually usher in the disease. These are: Coated tongue, anorexia, fetor of breath, epigastric discomfort, vomiting, and perhaps diarrhea. (2) Obstructive jaundice, indicated by yellow skin and conjunctivæ, light stools, and dark urine, is a constant symptom. (3) In some cases there is slight fever with swelling and tenderness of the liver.

Diagnosis.—This is based upon the acute course, the mild character of the symptoms, the history of preceding gastric catarrh, and the youth of the patient.

Prognosis.—This is, as a rule, favorable. The average duration of the disease is from two to six weeks.

Treatment.—The diet should be simple and digestible. Fatty and saccharine foods should be avoided. Milk, broths, eggs, lean meats, oysters, and well-cooked cereals are admissible. Sodium phosphate (1 dram three times a day), silver nitrate (1/4 grain three times a day), and ammonium chlorid (5 to 10 grains three times a day) are of value in relieving the primary gastro-duodenal catarrh. In obstinate cases nitrohydrochloric acid may prove beneficial. Daily irrigation of the colon with from 1 to 2 quarts of cold water is sometimes of service. Free water-drinking between meals is to be recommended. Alkaline mineral waters (Vichy, Vals, Hathorn) often act well.

Chronic catarrhal jaundice may follow repeated acute attacks, but in the large majority of cases it is a sequel of stenosis of the common bile-duct from gall-stones, stricture, or pressure from without. A constant symptom is chronic jaundice. Jaundice developing gradually without pain and increasing steadily from week to week, while the gall-bladder increases in size, is suggestive of *compression of the common* *duct by a tumor or cicatricial tissue;* persistent jaundice of varying intensity, preceded by colicky pains, and accompanied by ague-like paroxysms of a fever chill and sweat, is suggestive of *obstruction of the common duct by gall-stone.*

SUPPURATIVE CHOLANGITIS

Etiology.—Suppurative inflammation of the biliary ducts is usually a sequel of gall-stones or of obstruction of the ducts by tumor. Occasionally it follows a general infection, such as typhoid fever or pneumonia. The chief provoking organisms are the pyogenic cocci, the colon bacillus, and the typhoid bacillus.

Symptoms.—These comprise the usual phenomena of sepsis (fever, chills, sweats, and leukocytosis), with jaundice, local discomfort or actual pain, enlargement of the liver, perhaps also of the spleen, and emaciation. The gall-bladder is generally distended from concomitant cholecystitis.

The disease is a grave one, and surgical intervention offers the only chance of cure.

ACUTE CHOLECYSTITIS

Definition.—Acute inflammation of the gall-bladder.

Etiology.—The disease is always infectious, the organisms most commonly present being the colon bacillus, typhoid bacillus, pneumococcus, staphylococcus, and streptococcus. Injury to the mucosa by gall-stones is an important predisposing factor. It is not an uncommon complication of infectious fevers, especially typhoid fever.

Pathology.—The inflammation may be catarrhal or suppurative. Suppurative cholecystitis (*empyema of the gall-bladder*) is usually associated with purulent inflammation of the bile-ducts, and, unless promptly relieved by operation, proceeds to ulceration or gangrene and general peritonitis.

Symptoms.—In catarrhal cases the symptoms are slight fever, pain in the hepatic region, tenderness and enlargement of the gall-bladder, and, occasionally, jaundice. In the suppurative form there are severe paroxysmal pain, vomiting, a septic type of fever, leukocytosis, enlargement and tenderness of the gall-bladder, and, in some cases, jaundice.

Diagnosis.—It must be distinguished from *appendicitis, subphrenic abscess*, and *acute pancreatitis*. The discriminating features are the history of previous cholelithiasis, typhoid fever, or pneumonia, and the locality of the pain, tenderness, and swelling.

Prognosis.—Catarrhal cholecystitis usually subsides in from one to three weeks, but gall-stones and adhesions between the gall-bladder and adjacent organs are common sequels. In suppurative cases operation affords a reasonable hope of benefit.

CHOLELITHIASIS

(Gall-stones; Biliary Calculi)

Etiology.—Gall-stones are three or four times more common in women than in men. They occur most frequently after middle life, and are rarely seen before twenty-five. Sedentary habits, high living, tight lacing, obstruction of the ducts, and other factors that favor stagnation and inspissation of the bile predispose to their formation. Their occurrence after typhoid fever and other infections is especially common. The immediate cause of cholelithiasis seems to be a mild catarrhal inflammation of the biliary tract set up chiefly by typhoid or colon bacilli. This catarrh leads to the increased formation of cholesterin and to the precipitation of bilirubin-calcium. The latter, according to Naunyn, is the cementing substance which binds together the cholesterin, desquamated epithelium, agglutinated bacilli, etc.

Pathology.—Gall-stones may be found in the ducts, but in the large majority of cases they originate in the gall-bladder. There may be one or several hundred. When multiple, they are found with facets, from attrition. The size varies from that of a grain of sand to that of a large walnut. The color varies from light yellow to dark green. The chief constituent is cholesterin, but bile-pigments and lime-salts may also enter into their composition. On section, they usually present a concentric arrangement.

Events.—(1) Gall-stones may remain quiescent or give rise to no symptoms other than those of continued or recurring indigestion, with ill-defined pains in the upper part of the abdomen. (2) In consequence of violent expulsive efforts, excited by irritation of the gall-bladder, they may be extruded into the bowel, intense pain (*biliary colic*) marking their passage through the ducts. (3) Instead of making a complete exit, they may slip back into the gall-bladder or they may become impacted in the cystic duct, or, more often, in the lower part of the common duct. (4) They may perforate into the duodenum, peritoneum, lung, stomach, or kidney, or externally. Perforation may be followed by stricture of the ducts or by fistulous communications between the ducts and adjacent viscera. Perforation into the duodenum is not a rare cause of intestinal obstruction. (6) Although gall-stones are the result of a mild catarrh of the gall-bladder, their presence favors fresh infection and, therefore, the occurrence of active, even suppurative, cholecystitis or cholangitis. From the gall-bladder the inflammation may spread to the peritoneum, and if acute, result in a circumscribed abscess, or if chronic, lead to the formation of adhesions, which in turn may occasion digestive disturbances and colicky pains or even give rise to gastrectasis or intestinal obstruction. (7) The prolonged irritation excited by calculi may ultimately lead to carcinoma of the biliary passages.

Symptoms of Biliary Colic.—(1) The attacks begin abruptly with intense pain radiating from the hypochondriac region to the right shoulder. There are usually tenderness and rigidity over the gall-bladder. Chill and fever (102°–103° F.) often mark the onset. The symptoms of intense pain are obvious—anxious face, cold sweat, feeble pulse, and vomiting. Jaundice may follow from obstruction, but it is often absent.

If the stone escapes, it may subsequently be found in the stools. The attack may last from a few hours to several days.

Diagnosis.—*Renal Colic.*—In this affection the pain radiates from the lumbar region along the ureter into the bladder and genitals. Frequent micturition is a common symptom. There is no jaundice. Blood or the stone may be found in the urine.

Intestinal colic produces pain that radiates around the umbilicus. There are flatulence and borborygmi. Jaundice is absent.

Gastralgia.—Pain is over the whole stomach, does not radiate to the shoulder, and is relieved by pressure. There is no jaundice.

Gastric Ulcer.—Pain is closely related to eating. There are localized tenderness in the epigastrium, hyperacidity, and frequently hematemesis.

Symptoms of Obstruction of the Cystic Duct.—Obstruction of the cystic duct may be followed by cholecystitis (catarrhal, suppurative, or gangrenous), by atrophy of the gall-bladder, or by dropsy of the gall-bladder (*hydrops vesicæ felleæ*). In the last condition the gall-bladder can often be felt as a pear-shaped, elastic, movable tumor, projecting from the lower margin of the liver. Jaundice is not present, and subjective symptoms are slight.

Symptoms of Obstruction of the Common Duct.—In typical cases the symptoms are—(1) Chronic jaundice showing marked variations in intensity; (2) pain, also subject to distinct exacerbations; (3) recurrent attacks of intermittent fever, with chills and sweats (Charcot's hepatic fever). The liver is not enlarged; the gall-bladder is usually not distended, but often atrophied from antecedent attacks of cholecystitis (Courvoisier's law). Obstruction of the common duct may persist for months or years. It not infrequently leads to suppurative angiocholitis, to obstructive biliary cirrhosis, or to acute or chronic pancreatitis.

very promptly to a few whiffs of chloroform. In the mild but rather persistent attacks a few doses of antipyrin in hot water may suffice. The external application of heat (poultice or hot bath) is very useful.

When vomiting is urgent, carbonated water or champagne may be given. In threatened collapse diffusible stimulants are needed.

Obstruction of the Common Duct.—The measures best suited for promoting the advance of the stone into the bowel are rest, regulation of diet, the free use of alkaline mineral waters, the occasional exhibition of saline laxatives, and the application of heat to the hypochondriac region. Olive oil has been recommended as a special remedy, but it is of doubtful efficacy. As the sequelæ of impaction of the common duct are so grave, surgical aid should be invoked if the obstruction is not removed under medical treatment within a period of three or four weeks.

HYPEREMIA OF THE LIVER

Varieties.—(1) Active hyperemia. (2) Passive hyperemia.

Etiology.—*Active hyperemia* is commonly due to dietetic indiscretions. It may result from overindulgence in alcohol. It is often present in the infectious fevers. It appears to arise idiopathically in hot climates.

Passive hyperemia results from diseases that obstruct the venous circulation, as chronic heart and lung disease.

Pathology.—The liver is enlarged and filled with blood. In the *passive* variety, the center of the lobule, the area of the hepatic vein, is deeply pigmented, while the periphery, the area of the portal vein, is pale. This mottled appearance has given rise to the term "nutmeg liver." In persistent cases pigmentation, atrophy of the liver-cells, and overgrowth of the connective tissue result—a condition termed "cyanotic induration."

Symptoms.—*Active Hyperemia.*—The liver is enlarged and somewhat tender. There is a sense of fulness or even actual pain in the hepatic region. There may be slight jaundice. Digestive disturbances—anorexia, nausea, flatulence, headache, and epigastric tenderness usually coexist.

In the *passive variety* the symptoms are much the same as in the active form, but in addition there is frequently ascites. The liver is often quite large, and in extreme cases, such as follow tricuspid regurgitation, it may pulsate.

Prognosis.—In simple active congestion the prognosis is good. In passive congestion the prognosis depends upon the cause.

Treatment.—Active hyperemia from dietetic errors usually yields promptly to restriction of the diet and the administration of a mercurial purge, followed by a saline—Rochelle salt, Seidlitz powder, or sodium phosphate. In recurring attacks, in addition to hygienic and dietetic regulations, a pill like the following often proves useful:

℞. Massæ hydrargyri		gr. v
Pulveris rhei		
Extracti gentianæ	āā	ʒss
Olei caryophylli		gtt. iv.—M.

Fiant pilulæ No. xx.

SIG.—One or two occasionally, as directed; to be continued, if required, thrice daily for several days.

In passive congestion treatment must be directed to the primary disease. In mild cases alkaline mineral waters (Carlsbad, Congress, and Friederichshall) do well. A mercurial laxative may be used from time to time. In severe cases the most effective measures are absolute rest, a milk diet, saline purges, and wet-cupping over the liver.

CIRRHOSIS OF THE LIVER

(Chronic Interstitial Hepatitis)

Definition.—A chronic disease of the liver characterized by a hyperplasia of the connective tissue and more or less extensive retrograde changes in the liver-cells.

Varieties.—The most important varieties are *atrophic* or *portal cirrhosis*, *hypertrophic biliary cirrhosis*, *syphilitic cirrhosis*, and *capsular cirrhosis*.

ATROPHIC CIRRHOSIS

(Laennec's Cirrhosis; Alcoholic Cirrhosis; Gin-drinker's Liver)

Etiology.—It occurs most commonly in males of middle age. The chief cause is the continued use of alcohol, especially in the form of raw spirits. It has been suggested that poisonous food products, bacterial toxins or even microorganisms themselves absorbed from the intestines may also produce this disease. It is possible that some cases owe their origin to the specific fevers.

Pathology.—The liver may be very small, but in many cases it is considerably larger than normal. It is dense and inelastic. The surface is often studded with numerous roundish elevations ("hobnails"). The cut surface, which is usually yellowish in color, displays a network of fine and coarse pearly bands of connective tissue.

Microscopic examination reveals an increased amount of connective tissue, commonly of a fibrous character and chiefly interlobular or periportal in distribution. The parenchymatous cells, especially those about the periphery of the lobules, show various degenerative changes, and in advanced cases marked atrophy. On the contrary, evidences of proliferation of the liver-cells, as well as of the bile-ducts, are usually found, even in late stages of the disease. It is probable that destruction of certain liver cells through the activity of the poison is the primary change, and that the fibrosis and proliferation of the liver cells are secondary features.

Symptoms.—Obstruction of the portal circulation first causes *congestion and catarrh of the stomach*, hence the initial symptoms are anorexia, fetor of the breath, fulness and distress after eating, eructations, nausea, vomiting of mucus, flatulence, and constipation. For months and even years is muddy in appearance [illegible] common and occurs only as a [illegible] iptoms—delirium, stupor, [illegible] and [illegible]—usually appear toward the end of the disease. They are probably due to the retention of poisons that the liver is unable to convert or to eliminate.

he majority of cases terminate fatally in from [illegible] years, or in from one to two years after the [illegible] y circulation fails. Death results from exhaustion, [illegible] ge, pulmonary edema, intercurrent disease, [illegible]

omplications.—The kidneys, heart and [illegible] often coincidentally involved in the [illegible] berculosis, especially of the peritoneum, is a [illegible] n complication.

Diagnosis.—In the early stage the diagnosis [illegible] suspected. In the [illegible] th enlargement of the liver [illegible] ease.

Thrombosis of the portal vein produces a similar [illegible]

Splenic Anemia.—This disease is chiefly distinguished by the absence of a history of alcoholic excess, the early appearance and marked degree of the splenic enlargement, and the very pronounced anemia.

Chronic Perihepatitis.—In this disease the etiologic factors of cirrhosis of the liver are absent, the course is slow, the ascites often remains stationary for long periods and returns again and again after tapping, and signs of marked portal obstruction (abdominal varices and hemorrhages) are lacking.

Prognosis.—The outlook for permanent relief is bad.

Treatment.—Alcohol must be interdicted. A diet of bland, readily digested food is indicated. The gastric catarrh should receive appropriate treatment. Lavage of the stomach is contraindicated on account of the presence of esophageal varicosities. Potassium iodid is of service in syphilitic cases, but not otherwise. Ammonium chlorid (10 grains three times a day) is sometimes useful. Portal congestion is best relieved by the administration of salines (sodium phosphate or Rochelle salt) in hot water one-half hour before breakfast.

Ascites can sometimes be removed by the administration of cathartics and diuretics. A concentrated solution of Epsom salts (½ to 1 ounce), taken in the morning before breakfast, is usually the most efficient purgative. Occasionally it may be desirable to substitute compound jalap powder or elaterium. The diuretics of approved value are potassium acetate or bitartrate, digitalis, and squills.

Guy's or Baillie's pill has a well-deserved reputation:

℞. Massæ hydrargyri
Pulveris digitalis
Pulveris scillæ . . . . āā gr. xx.—M.
Fiant pilulæ No. xx.
Sig.—One pill thrice daily.

When the ascites is large and does not yield readily to drugs, paracentesis should be practised (see p. 120).

Surgical Treatment.—Talma's operation (suture of the omentum to the margin of the abdominal incision and irritation of the peritoneal surfaces of the liver) or one of its modifications, has proved of some benefit in a limited number of cases of liver cirrhosis with ascites. The object of the operation is to establish a compensatory circulation by making accidental adhesions and thus increasing the anastomoses between the vessels of the portal system and those of the systemic circulation. The operation is contraindicated if cardiac or renal disease coexists.

HYPERTROPHIC BILIARY CIRRHOSIS

(Hypertrophic Cirrhosis of Hanot)

Etiology.—The causes of hypertrophic biliary cirrhosis are obscure. Alcoholism does not appear to be a factor. An infective origin has been suggested. The disease is seen chiefly in men between twenty and thirty-five years of age.

Pathology.—The liver is greatly enlarged throughout the entire course of the disease. The organ is of a yellowish or greenish color, and its surface is smooth or finely granular. The spleen is enlarged.

Microscopic examination of the liver reveals an overgrowth of connective tissue, but this is delicate and fibrillary, and shows a marked tendency to enter the lobules and form a fine network between the cells (intralobular or monolobular cirrhosis). The small bile-ducts show proliferation of their epithelium and often contain plugs of inspissated bile. Many so-called new bile-ducts are seen. The liver cells often show comparatively little change. The chief features distinguishing it from atrophic or portal cirrhosis are the *intralobular* distribution of the fibrosis, the inflammatory changes in the biliary capillaries (radicular cholangitis) and the inconspicuous damage to the liver cells.

Symptoms.—The liver is uniformly enlarged, usually to a marked degree, throughout the course of the disease. Not rarely it is slightly tender. The spleen is also enlarged. Jaundice, varying in degree from time to time, is a conspicuous feature. Periodic attacks (crises) of pain in the hepatic region, attended by fever, leukocytosis, increase of jaundice, and, perhaps, vomiting, are of common occurrence. Toward the end of the disease hemorrhages into the skin and from the mucous membranes and symptoms of hepatic intoxication, such as delirium and stupor, often develop. Ascites, profuse hematemesis and enlargement of the superficial abdominal veins are rarely observed. The prognosis is unfavorable, but the course of the disease is long—often from five to ten years.

Treatment.—The treatment is in the main that suggested for hyperemia of the liver (see p. 103). In a number of instances the gall-bladder has been drained with good results.

OTHER FORMS OF CIRRHOSIS OF THE LIVER

Syphilitic Cirrhosis of the Liver.—In the diffuse form the appearance of the liver is similar to that of alcoholic cirrhosis. In the gummatous form, however, the organ is enlarged and often coarsely lobulated from fibrous transformation of the gummata. Other manifestations of syphilis, a positive Wassermann reaction, an enlargement of the liver that is grossly nodular, and the presence of unexplained fever should suggest the application of the therapeutic test.

Capsular Cirrhosis (Chronic Perihepatitis, Zuckergussleber).—This form is characterized by enormous thickening of the capsule of the liver (sugar-iced liver). The symptoms closely resemble those of atrophic cirrhosis, but the course is extremely slow, the ascites remaining stationary for long periods and returning again and again after tapping. In many cases chronic capsulitis of the spleen, chronic pleurisy, and chronic obliterative pericarditis are also present, constituting the disorder known as multiple serositis, Pick's disease or Concato's disease.

ABSCESS OF THE LIVER

(Acute Suppurative Hepatitis)

Etiology.—Abscess of the liver is always due to the action micro-organisms—Amœba coli, streptococcus, staphylo-:cus, colon bacillus. They may enter the liver directly from adjacent organ or through the portal vein, hepatic artery, e-ducts, or lymphatics.

(1) Amebic dysentery is a very common cause, the amebæ tering the liver through the portal vein. Occasionally iebic abscesses occur without any evidence of dysentery. ı Septic emboli from gastric ulcers, duodenal ulcers, purulent pendicitis, etc., may also lodge in the branches of the portal in and thus excite a suppurative inflammation (suppurative lephlebitis). (3) Pyogenic organisms may enter the patic artery in ulcerative endocarditis, abscess of the ıgs, and general pyemia. (4) Suppuration by way of the e-ducts sometimes occurs in angiocholitis secondary to ll-stones. (5) Traumatism may be a causal factor. (6) casionally gastric ulcer, ulcerative cholecystitis, perinephric scess, or empyema invades the liver and sets up suppuration side of it. (7) Finally, suppuration may result from the condary infection of an echinococcus cyst.

Pathology.—The abscesses following amebic dysentery tropical abscess") and traumatism are generally solitary, d usually occupy the right lobe. Metastatic abscesses e multiple.

Events.—Hepatic abscess may kill by septic poisoning or perforation into the lung, peritoneum, stomach, pleura, ricardium, or vena cava. Recovery may follow operation spontaneous rupture into the bronchi, into the stomach bowel, or externally.

Symptoms.—*Local Symptoms.*—The liver is enlarged and nder. The enlargement is more often upward than downard. Circumscribed bulging beneath the costal arch is metimes noted. Fluctuation is occasionally detected.

There is usually severe pain in the liver region and right shoulder. Exploratory puncture may reveal pus. Slight jaundice may develop, but it is often absent.

Constitutional Symptoms.—These result from sepsis, and include fever of a remittent or irregular type, chills, profuse sweating, marked anemia, and leukocytosis.

Rupture into the lung is characterized by severe cough, weak breath sounds at the base of the right lung, and the expectoration of large amounts of pus, often mixed with blood. In the case of amebic abscess the sputum may have a chocolate color and may contain amebæ.

Diagnosis.—*Hydatid cysts* develop slowly, are not painful, are not associated with septic phenomena, and yield clear fluid on aspiration.

Cancer of the Liver.—The history, marked cachexia, involvement of other organs, presence of jaundice, detection of hard nodules on the surface of the liver, and the absence of septic phenomena will suggest cancer.

Intermittent Fever due to Impacted Gall-stones.—In this condition the pain, fever, and sweating are often periodic; the health in the intervals may be well preserved; the jaundice increases at each paroxysm; the symptoms may persist for several years.

Prognosis.—Embolic abscesses are invariably fatal. Traumatic and amebic abscesses may terminate favorably upon spontaneous or induced evacuation.

Treatment.—In multiple abscesses treatment is palliative. Large solitary abscesses should be opened and drained.

CANCER OF THE LIVER

Etiology.—Cancer of the liver is more common in men than in women. It is infrequent before the age of forty. Heredity, traumatism, and chronic irritation from gall-stones are predisposing factors.

Pathology.—Primary cancer of the liver is rare; secondary cancer is common. The primary form may appear as a single large nodule (*massive cancer*) or as a widespread infiltrating growth (*nodular cancer*). The latter form is sometimes associated with cirrhosis of the liver (*cirrhotic cancer*). The secondary variety is usually due to the lodgement in the portal capillaries of cancerous emboli derived from a primary growth in one of the adjacent organs, especially the stomach. The liver is much enlarged, and studded with numerous grayish-white nodes, some of which project from the surface. The superficial nodes are often depressed at the center.

Symptoms.—(1) The liver is enlarged and painful, and often presents one or more smooth, hard nodules. The latter may show a central depression. (2) Cachexia is pronounced and develops rapidly. (3) Jaundice is common, but it is rarely intense. (4) Digestive disturbances are a prominent feature, and often precede the hepatic symptoms. Ascites sometimes results from portal obstruction. Toward the end, slight fever, delirium, stupor, and coma may develop (hepatic intoxication).

Diagnosis.—*Hypertrophic biliary cirrhosis* may be distinguished by the smooth, uniform enlargement of the liver, the enlargement of the spleen, the persistence of icterus without loss of color in the stools, the absence of marked cachexia, the age of the patient (between twenty and forty), and the slow course.

Abscess.—This may be distinguished by the history, the septic fever, and the results of exploratory puncture.

Syphilis of the Liver.—Other manifestations of syphilis, a positive Wassermann reaction, and the therapeutic test are the important aids in diagnosis.

Prognosis and Treatment.—The outlook is hopeless. The duration is from a few months to a year. Treatment can only be palliative.

HYDATID CYST OF THE LIVER

(Echinococcus of the Liver)

Etiology and Pathology.—Hydatid cysts are formed by the embryos of the Tænia echinococcus, a small tapeworm inhabiting the intestines of the dog. The disease is common in Iceland, Australia, and some parts of Europe, but is rare in America.

The eggs of the worm are accidentally ingested by man, and embryos are liberated in the stomach, whence they may migrate to any organ; the liver, however, is most commonly affected through the portal vein. The fixed embryo soon develops into a cyst that is composed of an external laminated layer and an internal breeding layer. A connective-tissue layer is formed on the outside from irritation.

The cyst contains a clear, non-albuminous fluid of low specific gravity (1002 to 1010) and rich in sodium chlorid.

Scolices or larvæ develop from the breeding layer; they are provided with four suckers and a circle of hooklets, and produce daughter-cysts within the parent-cyst. When ingested by the dog, the larvæ develop into mature tapeworms.

Symptoms.—Small cysts excite no symptoms. Large cysts produce an irregular enlargement of the liver, with a sensation of weight or fulness in the hypochondriac region. If the cyst is superficial, an elastic, fluctuating tumor may be detected on palpation. On percussion a peculiar vibratory sensation (hydatid thrill) may be imparted to the hand. Aspiration yields a clear fluid containing the characteristic hooklets. Fever, pain, and jaundice are usually absent. An increase of the eosinophiles of the blood (5 to 20 per cent.) is the rule.

Events.—(1) The cyst may reach a certain size and then become quiescent. (2) Trifling injury may convert it into abscess. (3) Rupture of the cyst into adjacent organs may terminate in death or in recovery.

Diagnosis.—The diagnostic features are a smooth, tense, elastic tumor of the liver, of slow growth, without pain, fever,

Prognosis and Treatment.—The prognosis depends somewhat upon the curability of the primary disease. The outlook, however, is always grave. The treatment must be directed to the causal disease.

ACUTE YELLOW ATROPHY OF THE LIVER

(Acute Parenchymatous Hepatitis; Icterus Gravis)

Definition.—A very rare and grave disease characterized anatomically by a rapid destruction of the liver tissue, and manifested clinically by jaundice, hemorrhages, a reduction in the size of the liver, and marked cerebral phenomena.

Etiology.—The disease occurs more frequently in women than in men. It is usually seen between the ages of twenty and thirty. Pregnancy is the most important predisposing factor. Alcoholic excesses, syphilis, and emotional excitement are also thought to have some etiologic significance. Acute yellow atrophy of the liver appears to be closely related to severe cases of epidemic catarrhal jaundice and to cases in which acute degenerative changes are engrafted on some pre-existing disease of the liver, such as cirrhosis. The rapid course, widespread lesions, and the fact that the disease has occurred endemically suggest a toxic or infectious origin.

Pathology.—The liver is reduced in size, flaccid, and friable. The surface is yellowish red and mottled. Microscopic examination reveals extensive necrosis of the liver-cells, hemorrhagic extravasations, hematogenous pigmentation, and occasionally small-celled infiltration. The other organs are usually the seat of fatty and parenchymatous degeneration.

Symptoms.—(1) The initial symptoms are those of catarrhal *jaundice*. (2) Nervous symptoms (*cholemia*) soon follow; these are severe headache, maniacal delirium, stupor, and coma. (3) The urine is scanty, and usually contains *leucin* and *tyrosin*, bile, albumin, and tube-casts. The excretion of urea is often greatly diminished. (4) The area of hepatic percussion dulness rapidly decreases. (5) Hemorrhages from the mucous membranes and into the skin are common. Fever is usually absent. The disease rarely lasts longer than a week or ten days. Recovery is extremely rare.

Diagnosis.—In *acute phosphorus-poisoning* acute gastritis precedes the jaundice, the vomitus and stools may be phosphorescent or have the odor of phosphorus, the liver is usually enlarged, and the urine less frequently contains leucin and tyrosin.

In *hypertrophic cirrhosis* the liver is enlarged and often painful, the course is slow, and leucin and tyrosin rarely appear in the urine. In *Weil's disease* the liver is enlarged and high fever is a constant symptom.

Treatment.—This must be symptomatic.

WEIL'S DISEASE

This is a rare form of infective jaundice, occurring in epidemics and attacking most commonly males between the ages of fifteen and thirty. It is probably acquired by eating tainted meat or drinking contaminated water. Cloudy swelling and fatty degeneration of the cells of the liver and kidneys and focal necroses in the liver are the chief postmortem changes.

The characteristic symptoms are high fever (103°–104° F.), coming on suddenly with malaise and muscular pains; slight jaundice, enlargement of the liver and spleen, albuminuria and marked nervous disturbances (headache, dizziness, delirium and somnolence). Recovery usually ensues but convalescence may be slow and relapses are not uncommon. The disease appears to be related on the one hand to epidemic catarrhal jaundice and on the other to acute yellow atrophy of the liver.

ACUTE PERITONITIS

Definition.—An acute inflammation of the peritoneum. The process may be general or localized.

Etiology.—The disease is probably always caused by bacteria, which enter the peritoneum from the adjacent viscera, especially the alimentary canal, from the Fallopian tubes, from external wounds, or directly from the blood. The organisms most frequently found are the Streptococcus pyogenes, Staphylococcus pyogenes, Bacillus coli, pneumococcus, Bacillus pyocyaneus, and gonococcus.

Peritonitis may follow—(1) *Perforation* of the peritoneum by an external wound, by rupture of a gastric or intestinal ulcer, by rupture of a suppurating appendix, gall-bladder, or Fallopian tube, or by rupture of a visceral abscess; (2) *extension* of a septic process in adjacent structures—stomach, bowel, gall-bladder, pancreas, uterus, etc.; (3) *traumatism;* (4) *general infections*—septicemia, specific fevers, tuberculosis, etc.

Pathology.—The serous surfaces first become red and lusterless; later a serofibrinous, fibrinous, or purulent exudate is formed. Putrid and hemorrhagic exudates are sometimes observed.

Symptoms.—The most prominent symptoms are intense abdominal pain and tenderness. The breathing is shallow and thoracic. To relieve the tension of the abdominal muscles, the patient lies motionless upon his back, with the legs and thighs flexed. The features are pinched, and the expression is anxious. The abdomen is distended, and its walls are rigid. Percussion at first reveals general tympany, but later there may be dulness in the flanks from the gravitation of the exudate. The temperature is usually moderately high (102°–104° F.), and the pulse is small, rapid, and "wiry." The bowels are usually constipated. Vomiting and hiccup are common symptoms. In severe cases collapse speedily ensues, and is indicated by a fall in the temperature, a cold, clammy surface, a rapid feeble pulse, and suppression of urine.

In *localized* peritonitis the constitutional symptoms are less severe. Pain, tenderness, and rigidity are circumscribed. General tympanites is usually absent. Abscess formation is common.

Diagnosis.—*Acute Enteritis.*—In this disease the pain is colicky and less intense; tenderness is much less marked; rigidity is rarely present; there is diarrhea; the constitutional symptoms are not so grave.

Intestinal Obstruction.—Constipation is absolute; vomiting is stercoraceous; fever and abdominal tenderness are less pronounced.

Hysteric Abdomen.—This condition may closely resemble peritonitis. The personal history must be carefully considered. Fever is not usually present, the pulse is not usually rapid and wiry, and when the attention is distracted, the pain may disappear.

Prognosis.—The prognosis in acute diffuse peritonitis is always grave, and in perforative cases it is practically hopeless unless operative treatment is soon instituted. In localized peritonitis the outlook is much more favorable.

Treatment.—The patient should be kept in a semi-sitting (Fowler's) position, so that the exudation will flow toward the pelvis and away from the diaphragm. All food and liquids by the mouth should be withheld and thirst relieved by enemas of salt solution. Persistent vomiting is best treated by washing out the stomach. Collapse must be combated by the external application of heat, the subcutaneous administration of strychnin, and the intravenous injection of adrenalin in salt solution (1 in 50,000).

Surgical intervention is indicated in the great majority of cases. The operation should be done speedily, the exudation released, and the cause removed with the least possible trauma and handling of the parts. Free drainage should be maintained by Fowler's position. Irrigation of the abdominal cavity is probably better avoided, although some very able surgeons believe that it is often indicated. To maintain the proper degree of tension in the vessels and to favor the elimination of toxins, enteroclysis by the drop (Murphy's) method is invaluable. Morphin in large doses is harmful, but the entire withholding of it is rarely justifiable.

CHRONIC DIFFUSE PERITONITIS

Etiology.—Chronic peritonitis may be a sequel of acute peritonitis. In a few instances it has seemed to have resulted from syphilis. In the vast majority of cases it is tuberculous or cancerous.

Pathology.—The intestines are matted together by adhesions. The omentum is often contracted and greatly thickened. Effusion is usually present, but it varies considerably in amount; it is highly albuminous, and in the tuberculous and cancerous varieties it may be bloody.

Symptoms.—Fever is slight and may be absent. Pain is not severe, and is frequently paroxysmal. There is usually more or less diffuse tenderness. Anemia and emaciation are often pronounced. The abdomen is usually distended, often irregularly, from sacculated effusions, inflated intestinal coils, or the projecting matted omentum. Palpation often detects a friction fremitus and resistant masses or nodules. Percussion yields dulness, varying in extent with the amount of effusion. If the fluid is sacculated, the dulness may be irregularly distributed. Fluctuation can sometimes be elicited. On tapping, the fluid is turbid, rich in albumin, and of high specific gravity (about 1015). In cancerous and tuberculous cases it is frequently bloody.

Diagnosis.—The diagnosis between *tuberculous* and *cancerous* peritonitis is not always easy. The tuberculous form usually occurs in persons under forty, gives rise to less cachexia than the cancerous form, and is frequently associated with tuberculous foci elsewhere, especially in the lung, pleura, testis, or Fallopian tube. In doubtful cases the tuberculin test may be employed or a guinea-pig may be inoculated with the exudate.

Prognosis.—Cancerous peritonitis is invariably fatal. Tuberculous peritonitis, while always grave, not infrequently ends in recovery, especially in children.

Treatment.—In the tuberculous form the general treatment should be that of pulmonary tuberculosis. If the effusion is large, aspiration will be required. Surgical treatment (free incision with washing out of the abdominal cavity with normal salt solution) should be advised in suitable cases.

ASCITES

Definition.—A collection of serous fluid in the peritoneal cavity.

Etiology.—(1) It may result from the causes of general dropsy—heart disease, Bright's disease, chronic pulmonary emphysema, etc. (2) It is frequent in atrophic cirrhosis of the liver, and is sometimes seen in cancer and syphilis of the liver. (3) It is occasionally caused by thrombosis of the portal vein. (4) It is very common in the various forms of chronic peritonitis. (5) It is sometimes met with in tumors of the abdomen and in extreme enlargement of the spleen. (6) It is rarely due to obstruction or rupture of the thoracic duct (chylous ascites).

Symptoms.—If the infusion is large, a sensation of weight in the abdomen, dyspnea, scanty urination, and edema of the feet may result from pressure.

Physical Signs.—*Inspection.*—The abdomen is distended; the surface is smooth and shining; the base of the thorax is broadened; the navel is more or less obliterated; the superficial veins are frequently enlarged; and, when the patient lies in the dorsal position, the flanks bulge.

Palpation may elicit fluctuation, and in the flanks a sense of resistance.

Percussion reveals dulness and resistance in dependent parts, with superincumbent tympany. The dulness is movable and is detected in the flanks when the patient occupies the dorsal position.

Aspiration.—The fluid is usually clear, straw-colored, albuminous. The specific gravity is from 1010 to

A specific gravity above 1015 points to inflammation. A high percentage of endothelial cells suggests a mechanical causation; of polymorphonuclear leukocytes, subacute inflammation; of lymphocytes, tuberculous infection. In cancerous and tuberculous peritonitis the fluid is sometimes bloody. Occasionally, chylous fluid is present.

Diagnosis.—*Tympanites.*—This yields universal hyperresonance on percussion.

Ovarian Cysts.—The enlargement is at first unilateral. As the intestines are pushed aside, the dulness is anterior and the resonance is in the flanks. Vaginal examination often furnishes important data. The fluid of the cyst has a higher specific gravity (1025).

Distention of the Bladder.—The history, the location of the dulness, and the results of catheterization will make the diagnosis apparent.

Treatment.—Treatment should be directed to the original cause. Hydragogue cathartics and diuretics are sometimes useful. Concentrated saline solutions, compound jalap powder (20 to 40 grains), and elaterium (1/8 grain) are the most useful cathartics. Infusion of digitalis (3 to 4 fluiddrams), citrated caffein (2 to 3 grains), potassium citrate or acetate (20 grains), and Guy's pill (see p. 106) are the most reliable diuretics.

℞. Potassii citratis . . . . ℥ss
Infusi digitalis . . . . ℥vj.—M.
SIG.—A tablespoonful thrice daily.

If the effusion is large and causes discomfort or great diminution in the quantity of urine, paracentesis should be performed.

Paracentesis Abdominis.—The bladder having been emptied, the patient is placed in a semirecumbent position, and a spot in the median line midway between the umbilicus and the symphysis pubis is anesthetized by means of a block of ice sprinkled with salt. A stout trocar is now introduced with a quick thrust into the abdominal cavity, a rubber tube is attached to the cannula for the purpose of conveying the fluid into a pail placed below the patient's bed, and the trocar is then withdrawn. While the fluid is escaping, a many-tailed bandage is adjusted to the abdomen and gradually tightened. The application of such a binder should never be omitted. It gives support to the relaxed abdominal walls, and tends to prevent syncope and hematemesis. When the fluid ceases to flow, the cannula is removed, and the opening sealed with an antiseptic pad and a few strips of adhesive plaster.

In tuberculosis peritonitis, and in cirrhosis of the liver with recurrent ascites, surgical treatment sometimes proves successful.

Embolism and Thrombosis of the Mesenteric Vessels.—Occlusion of the mesenteric vessels occurs most frequently in the latter half of life. It may involve the arteries, the veins, or both, and is most commonly due to embolism originating in acute or chronic endocarditis, but it occasionally results from thrombosis due to sclerotic changes in the mesenteric artery or aorta, or to inflammatory changes in the mesenteric vein, occurring in association with morbid processes in the adjacent viscera. Hemorrhagic infarction of the intestine is the usual result. The chief symptoms are acute colicky abdominal pain, vomiting, profuse blood diarrhea, or constipation, abdominal distention, and shock. Peritonitis frequently supervenes. Occlusion of large vessels almost always ends fatally in a few days, unless operation with resection of the bowel is undertaken at an early stage.

DISEASES OF THE KIDNEYS

THE URINE

Normal urine is a pale, amber-colored fluid, of acid reaction, having a specific gravity of 1015 to 1025, and amounting in quantity to about 50 ounces (1500 c.c.) in twenty-four hours.

Polyuria.—An increased flow of urine.

Temporary polyuria may result from—(1) Excessive ingestion of fluids. (2) Administration of diuretics. (3) Suppression of perspiration. (4) Crises of certain febrile diseases, and certain neurotic manifestations, such as neuralgia and hysteria. (5) Absorption of serous effusions and transudations. (6) Removal of some temporary obstruction in the urinary passages.

Persistent polyuria may result from—(1) Diabetes mellitus. (2) Diabetes insipidus. (3) Chronic interstitial nephritis. (4) Amyloid kidney.

Oliguria, diminution in the quantity of urine, occurs in febrile disorders in conditions causing profuse sweating or diarrhea, in all conditions interfering with the flow of blood through the kidneys, such as valvular heart disease, pulmonary lesions, ascites, etc., and in certain inflammatory or destructive diseases of the kidneys themselves. **Anuria,** or suppression of urine, may occur (1) in acute nephritis and also sometimes in chronic nephritis; (2) after abdominal operations or injuries; (3) in collapse; (4) in hysteria; and (5) as a result of the mechanical obstruction of the urinary passages by tumors, calculi, enlarged prostate gland, etc.

Urea Excretion.—Normal urine contains about 2 per cent. of urea. The daily output varies considerably, depending upon the diet and other factors. The average amount is between 20 and 40 grams. Estimations of the urea excretion are of little value unless the total nitrogen intake and the total nitrogen output are also taken into consideration. The quantity of urea is diminished in starvation, in wasting diseases, after prolonged vomiting, and in diseases destroying the liver substance, as acute yellow atrophy. The total excretion of urea is also reduced, though the percentage is high, in acute and chronic parenchymatous nephritis. In chronic interstitial nephritis, until a late period, the total excretion of urea may be normal.

Lithuria.—The amount of uric acid excreted daily in the urine by healthy individuals varies from 0.2 to 1.25 grams, and the ratio of uric acid to urea in health is usually about 1 to 60 or 1 to 70. The uric acid is partly *endogenous*, or derived from the breaking down of the nuclei of the body cells, and partly *exogenous*, derived from the catabolism of the purin bodies of the food (meats, especially glandular organs, caffein from coffee, etc.). The amount of endogenous uric acid varies in different individuals, but it remains fairly constant for the same individual. A deposit of urates in the urine is no indication of an excess in the daily excretion. Such a deposit may result from increased concentration of the urine, a high degree of acidity, a deficiency of pigment (Klemperer), and even a low atmospheric temperature. Clinically, uratic deposits are common in febrile diseases, in various affections of the stomach and liver, in venous stasis of the organs from diseases of the heart or lungs, in leukemia, and in gout during and after the attack. Observations on uric acid excretion are of little value unless exact quantitative estimations of the daily totals are made and the nature and amount of the foods ingested are taken into consideration.

A sediment of *uric acid* has a reddish-brown color and resembles grains of cayenne pepper. Microscopically, uric acid appears as reddish-yellow rhombic prisms or lozenge-shaped crystals. Amorphous *urates* form a pinkish sediment having the appearance of brick dust, and microscopically occ as dark opaque granules. Occasionally urates occur in crystalline form, appearing as needles, dumb-bells, or globules with spiny processes.

Leucinuria and Tyrosinuria.—Leucin and tyrosin are found in the urine chiefly in acute yellow atrophy of the liver and phosphorus-poisoning, and, in small quantities, occasionally in the acute infectious diseases.

They may be detected by evaporating a few drops of the urine on a glass slide. Leucin appears in the form of small, round, glistening spheres, resembling fat-drops, but, unlike the latter, they are insoluble in ether. Tyrosin appears in the form of intersecting tufts of fine acicular crystals.

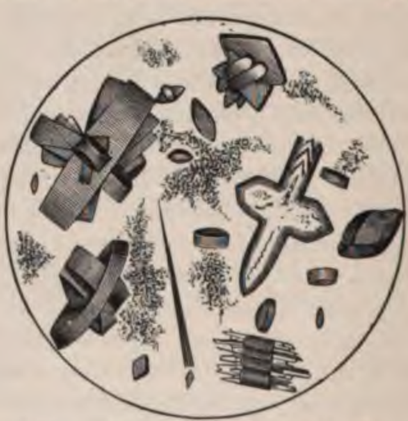

FIG 2.—Uric acid and uric acid salts.

Cystinuria.—Cystin rarely appears in the urine in consequence of an hereditary vice of metabolism, the patient being unable to complete the oxidation of the cystin normally formed in the disintegration of protein. It forms colorless hexagonal crystals freely soluble in ammonia and mineral acids. It is of clinical importance only in that it is prone to precipitate and form renal or vesical calculi.

Alkaptonuria.—This very rare condition results from a congenital inability to complete the combustion of the aromatic fractions of proteins. The urine contains an aromatic acid, homogentisic acid, and in consequence turns black on exposure to air, stains the clothing and reduces Fehling's solution. Alkaptonuria is harmless but occasionally leads to blackening of the cartilages, fibrous tissue and skin (*ochronosis*).

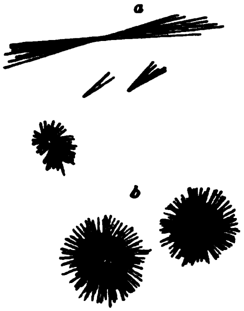

FIG. 3.—*a*, Tyrosin crystals; *b*, leucin crystals.

Phosphaturia.—Phosphates forming urinary sediments occur in urine that is alkaline, neutral or feebly acid. Both amorphous and crystalline forms are observed. The precipitation of *amorphous earthy phosphates* (calcium and magnesium) may be effected by warming the urine and adding to it a few drops of liquor ammoniæ. The precipitate resembles one of albumin, but is distinguished by being soluble in acetic acid. *Crystals of calcium phosphate* are observed in urine that is

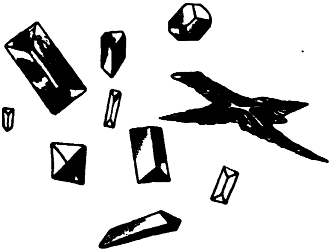

FIG. 4.—Triple phosphate.

still feebly acid, but which is about to undergo alkaline fermentation. The crystals are stellate or rod-shaped and are soluble in acetic acid.

Ammoniomagnesium phosphate, or triple phosphate, is formed in decomposing urine and appears as transparent coffin-shaped prisms or as feathery stellate forms. The shorter triangular prisms resemble crystals of calcium oxalate but, unlike the latter, are freely soluble in acetic acid. The detection of triple phosphates in freshly voided urine indicates decomposition in the bladder, the result of cystitis.

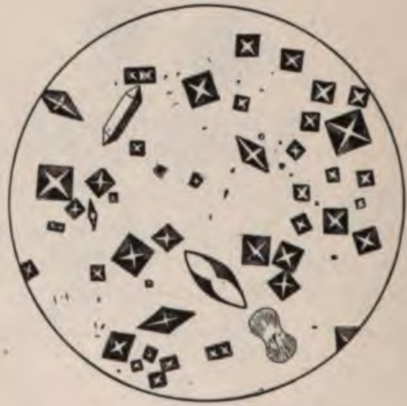

FIG. 5.—Oxalate of lime.

A deposition of phosphates in the urine does not in itself constitute evidence of excessive elimination. Such a deposition is often due to diminution of the acidity of the urine from various causes. An actual excess of phosphates can be determined only by quantitative methods. Clinically, increase of phosphates occurs in osteomalacia, diabetes, tuberculosis, and many other wasting diseases. It is not rarely associated with neurasthenia, but whether the relation is one of cause or effect is not known. In certain cases, especially in children, it seems to be the result of some disturbance of calcium metabolism. Finally, cases of polyuria with phosphaturia, resembling diabetes mellitus, have been described (*diabetes phosphaticus*).

Chlorids.—The quantity of these salts in the urine depends largely upon the amount of chlorids in the food. Clinically, the chlorids are diminished in febrile diseases, more especially in pneumonia. In cases of nephritis the retention of chlorids in the tissues constitutes one factor in the causation of dropsy. The quantity of chlorids may be roughly estimated by removing any albumin that may be present, then adding a few drops of a solution of silver nitrate and noting the amount of precipitate obtained.

Oxaluria.—Oxalates are deposited in the urine in the form of calcium oxalate which appears as minute, highly refracting octahedra or as dumb-bells. The amount of oxalate sediment bears no relation to the amount of oxalic acid excreted. Oxalate crystals are especially abundant after certain articles of diet (rhubarb, spinach and other vegetables), and in certain cases of indigestion marked by pronounced neurasthenic symptoms and mental depression.

Cylindruria.—This term is used to designate the occurrence of tube-casts in the urine. Tube casts are cylinders of albuminoid substances formed in the uriniferous tubules. They are often composed in part of epithelial cells, blood-cells, or the products of degenerated cells. They appear as:

Hyaline Casts.—These are clear, translucent cylinders, often so pale as to be scarcely visible. They occur in the urine in all forms of nephritis, in congestion of the kidneys, in jaundice, and even in health. They are frequently the only casts present in chronic interstitial nephritis.

Waxy Casts.—These resemble hyaline casts, but they appear more solid and rigid and are more or less yellow. They occur especially in chronic parenchymatous nephritis.

Epithelial Casts.—These are cylinders of epithelial cells or hyaline casts covered with epithelial cells. They occur especially in acute parenchymatous nephritis.

Granular Casts.—These are cylinders covered with the débris of broken-down epithelial cells. They may occur in any form of nephritis.

Fatty Casts.—These are casts studded with oil-drops derived from degenerated epithelium. They occur chiefly in chronic parenchymatous nephritis.

Blood Casts.—These are cylindric masses of red blood-cells, or, more commonly, hyaline casts studded with red blood-cells. They occur in acute and chronic hemorrhagic nephritis.

Pus and Bacterial Casts.—Casts composed respectively of masses of pus-cells and of bacteria are occasionally met with in suppurative nephritis.

Cylindroids.—These formations may resemble hyaline casts, but they are usually much longer and often taper off at one end to a thread. Moreover, they frequently show constrictions at different points. Their presence is not a proof of nephritis. They often occur in conditions of renal irritation.

Urobilinuria.—Urobilin is formed in the intestines by the action of bacteria on bilirubin. When present in excess, the urine is of a reddish-brown color. An increase of urobilin in the urine may occur—(1) In diseases associated with excessive destruction of red blood-cells, as pernicious anemia and scurvy; (2) after the absorption of hemorrhagic effusions; (3) in acute infectious diseases; (4) in certain liver diseases (cancer, cirrhosis, catarrhal jaundice). The yellowish pigmentation of the skin sometimes observed in cases of urobilinuria is due to bile pigment in the blood and not to the urobilin (see acholuric jaundice, p. 95).

Hematoporphyrinuria.—Hematoporphyrin is a product of the decomposition of hemoglobin. It is hematin deprived of its iron. Large amounts impart to the urine a dark red color. It is found in the urine in a large number of diseases, and in chronic poisoning by sulphonal and trional.

Glycosuria.—Grape-sugar in the urine.

observed—(1) In diabetes mellitus, due to lesions of the islands of Langerhans; (2) after the ingestion of large amounts of sugar (200 to 250 grams of grape-sugar); (3) in certain diseases of the pancreas and other glands having internal secretions, such as the hypophysis, thyroid and adrenals; (4) in injuries or diseases of the nervous system affecting directly or indirectly Claude Bernard's medullary center; (5) after the administration of phloridizin, the resulting glycosuria depending on changes in the renal cells (renal diabetes) and being unaccompanied by a hyperglycemia; (6) not rarely after the administration of ether or chloroform and in poisoning by morphin, anilin derivatives, cyanids, and many other drugs; (7) sometimes in pregnancy; (8) not rarely in the acute infections, grave anemias, and gout.

Qualitative Tests for Glucose.—The copper tests are commonly employed, and depend on the power of glucose in alkaline solution to reduce cupric hydroxid to cuprous hydroxid and cuprous oxid.

Fehling's Test.—As the fluid employed in this test spoils on keeping, it should be freshly prepared when required by mixing in equal proportions the following solutions:

First solution: Dissolve 34.64 grams of pure cupric sulphate in distilled water, and dilute up to 500 c.c.

Second solution: Dissolve 180 grams of pure Rochelle salt and 70 grams of caustic soda in 400 c.c. of distilled water, and heat to boiling; on cooling, make up to 500 c.c. with distilled water.

To about ten minims of each solution in a test-tube add about a fluidram of distilled water, and boil for a few seconds; if the solution remains clear, add the suspected urine drop by drop, and occasionally heat the tube. If sugar is abundant, a yellowish-red deposit will be produced. If no precipitate falls, continue the addition of the urine until an equal volume has been added, and allow to cool; then if no precipitate falls, sugar is absent.

It must be borne in mind that cupric hydroxid is sometimes reduced by substances in urine other than glucose, such as excess of uric acid or creatinin, alkapton, compound glycuronates following the administration of chloral, salol, turpentine, etc., and other sugars (lactose, pentose). In case of doubt the fermentation test should be tried.

The Phenylhydrazin Test.—Put in a test-tube half filled with water phenylhydrazin (hydrochlorate) 2 grains and sodium acetate 3 grains. Dissolve by heating. Fill the tube with suspected urine, and stand in boiling water for twenty minutes. Then place in cold water. On cooling, yellow, radiating groups of needle-shaped crystals of phenylglucosazon fall, which may be detected under the microscope.

Böttger's Test.—Add to a couple of drams of suspected urine which is free from albumin an equal volume of liquor potassæ and a few grains of subnitrate of bismuth, and boil; if sugar is present, it will reduce the salt of bismuth to black metallic bismuth. Substances containing sulphur, like albumin, yield a similar black precipitate.

The Fermentation Test.—Fill a four-ounce bottle three parts full of urine, and add a fluidram of ordinary yeast or a small portion of compressed yeast; lightly cork, and subject to a temperature of 70° to 80° F. for ten or twelve hours. If sugar is present, fermentation results with the evolution of carbon dioxid, and the specific gravity of the urine falls.

Quantitative Tests.—*Fermentation test*: Employ two bottles of urine, and to the one add the yeast; at the end of twenty-four hours take the specific gravity of each specimen. Every degree lost in the fermented urine indicates a grain of sugar to the fluidounce.

Fehling's Test.—To 1 c.c. of Fehling's solution add 4 c.c. of distilled water, and boil; if the solution still remains clear, add $1/10$ c.c. of the urine from a graduated pipet, and gently heat. Continue the addition of the urine, little by little, until all blue color has disappeared. If 1 c.c. of urine has been added, it will have contained half of 1 per cent. of sugar. If 2 c.c. are used, it will have contained ¼ per cent. If but ½ c.c. is used, it will have contained 1 per cent.

If the specific gravity indicates that the amount of sugar is great, dilute the urine with a definite amount of water, and estimate accordingly (Tyson).

Albuminuria.—Albumin in the urine.

Causes.—It occurs—(1) In all forms of nephritis; (2) in congestion of kidneys from diseases of the heart, lungs, and liver; (3) in conditions profoundly affecting the blood, as pernicious anemia, leukemia, purpura, and poisoning by many drugs; (4) in acute febrile diseases; (5) often in pregnancy; (6) occasionally in certain persons in health, as in young adults after exertion, after a cold bath, or even upon changing to the upright position (orthostatic albuminuria); (7) when the urine contains pus or blood (accidental albuminuria); (8) in many nervous diseases, as apoplexy, cerebral concussion, tetanus, epilepsy.

Tests for Albumin.—Heller's Test.—Pour a small quantity of colorless nitric acid in a test-tube, and allow an equal quantity of filtered urine to trickle from a pipet down the side of the tube and to come in contact with the acid. If albumin is present, a sharply defined white ring is formed at the line of junction.

Turpentine, copaiba, and other oleoresins eliminated in the urine yield similar rings, but the latter are redissolved on the addition of alcohol.

Uric acid produces an undefined pink ring, but it is not exactly at the line of contact, and is redissolved on the application of heat.

Bence-Jones protein produces a ring exactly like that of albumin with cold nitric acid.

Johnson's Test.—Fill a six-inch test-tube two-thirds full of filtered urine, and allow a couple of drams of a clear, saturated solution of picric acid to flow down the side of the tube and to mix with the urine. Turbidity indicates the presence of albumin, and it increases on gently heating the tube near its mouth. Certain substances in the urine, like the alkaloids, produce a similar turbidity, but this disappears on the application of heat.

Robert's Nitric Magnesium Test.—This test is very delicate and reliable. The test-fluid is made by adding one volume of strong nitric acid to five volumes of a saturated solution of sulphate of magnesium, and is employed in the same manner as nitric acid in Heller's test.

Albumosuria.—True albumose may be found in the urine when large amounts of protein are undergoing autolysis, as during postpartum involution of the uterus, in febrile diseases, in pneumonia during resolution of the exudate, and acute yellow atrophy of the liver. Albumose is not precipitated by heating after the addition of a few drops of acetic acid to the urine.

Bence-Jones protein, sometimes regarded as an albumose, appears very early in the urine in the neoplastic affection of the bone marrow known as multiple myeloma. Unlike ordinary albumin, it coagulates at a very low temperature (50°–60° C.), the coagulum redissolving on boiling and reappearing on cooling.

Acetonuria.—Acetone is probably derived chiefly from the fats through the intermediary stages first of β-oxybutyric acid and then of diacetic acid. The appearance of these bodies in abnormal amounts in the blood and urine (ketonuria) is spoken of as *acidosis.* Acetone occurs in the urine—(1) In diabetes mellitus; (2) in starvation, especially carbohydrate starvation; (3) in poisoning with chloroform and phosphorus; (4) in many acute infections; (5) sometimes in the pernicious vomiting of pregnancy; (6) in certain gastro-intestinal disturbances, especially of children. In the recurrent vomiting of children the digestive disturbances and the acidosis appear to be the result of a common cause.

Legal's Acetone Test.—To 4 c.c. of urine add a few drops of a strong, freshly prepared solution of sodium nitroprussid and a few drops of acetic acid (to prevent reaction with creatinin); then add sodium hydroxid solution. If acetone be present, the urine acquires a ruby-red color, which soon turns to a purplish red.

Diacetic acid and *beta-oxybutyric acid* are often found in the urine with acetone when the morbid processes responsible for the production of these compounds are very active. The presence of diacetic acid is indicated by the appearance of a Bordeaux-red color when a solution of ferric chlorid is slowly added to urine that has not been boiled. Salicylic compounds give the same reaction, but with these the color change is also observed in urine that has been previously boiled. There is no simple clinical test for beta-oxybutyric acid.

Hematuria.—Blood in the urine. The chief causal conditions are: (1) Traumatism; (2) acute inflammation of any part of the genito-urinary tract—kidneys, bladder, urethra; (3) calculi in the bladder or kidney; (4) congestion or infarction of the kidneys; (5) chronic interstitial nephritis (occasionally copious bleeding occurs); (6) certain general disorders affecting the blood, such as purpura hemorrhagica, scurvy, hemophilia, leukemia, malaria, etc.; (7) tumors or tubercle of the kidney or bladder; (8) varicose veins at the neck of the bladder (occasionally seen in old persons), enlargement of the prostate, benign or malignant; (9) parasites in the genito-urinary tract, such as the Filaria sanguinis hominis and Bilharzia hematobia.

Diagnosis.—Blood may be recognized by microscopic, spectroscopic or chemical examination. By the eye, blood may be confounded with bile, urobilin, or hematoporphyrin.

Heller's Test.—Boil the urine with a solution of caustic potash: phosphates are precipitated, which assume a red color from the freed hematin.

Source of Hemorrhage.—*Urethra.*—The urine first passed is bloody, and the other symptoms point to the urethra.

Bladder.—Bleeding often at the end of micturition and other symptoms point to the bladder.

Kidney.—The blood is intimately mixed. There may be blood-casts or clots, and the other symptoms point to the kidneys.

Hemoglobinuria.—Hemoglobin appears in the urine as a result of excessive hemolysis. The latter occurs under the following conditions: (1) In certain infections, notably malaria (black-water fever); (2) in poisoning by various drugs, such as potassium chlorate, coal-tar derivatives, etc.; (3) after severe burns; (4) after the transfusion of foreign blood; (5) sometimes in Raynaud's disease; (6) paroxysmal hemoglobinuria. In this last affection, which is sometimes of syphilitic origin, the attacks are usually precipitated by exposure to cold.

Indicanuria.—Indican, or potassium indoxyl sulphate, is a product of indol derived from the bacterial decomposition of proteids in the intestine. It does not color the urine, but by oxidation it is converted into indigo-blue. It is a constituent of normal urine. It is increased (1) in all conditions which favor putrefaction in the upper bowel, as obstruction in the small intestine, acute and chronic peritonitis, typhoid fever, intestinal catarrh, and obstructive jaundice; (2) conditions associated with the decomposition of pus, as empyema, abscess, and gangrene of the lung.

Tests for Indican.—Mix equal volumes of urine and fuming hydrochloric acid, and with constant shaking, add a fresh, saturated solution of calcium hypochlorite, drop by drop, until the blue color ceases to deepen, then shake with chloroform. The latter dissolves the indigo and separates as a blue liquid, the color of which is more or less deep, according to the amount of indican.

Choluria.—The presence of bile-pigments and bile acids in the urine. It is most marked in obstructive jaundice, but it may also occur in the non-obstructive form. The urine varies from a greenish-yellow to a dark brown color.

Tests for Bile.—Gmelin's Test.—Allow a few drops of urine and a few drops of fuming nitric acid to come together on a white plate. If bile is present, there will be an iridescent play of colors—green, blue, violet, and red—at the line of contact.

Pettenkofer's Test.—Add a few grains of cane-sugar and a drop of sulphuric acid to the suspected urine in a test-tube; heat gently, and if bile acids are present, a violet-red color is produced.

Test for Bile Acid.—Bile acids lessen the surface tension of urine, hence finely powdered sulphur sprinkled on the surface of the urine, instead of floating, sinks if bile acids are present (Hay's test).

Chyluria.—Chyle in the urine. The urine presents a milky appearance. The emulsion of the fat is so complete that microscopic examination rarely reveals distinct oil-globules. Ether dissolves the fat and renders the urine clear. Chylous urine is often slightly pink from the admixture of blood. Chyluria is usually due to obstruction of the lymphatic ducts by the Filaria sanguinis hominis, but a non-parasitic form of this condition, obscure in origin, is sometimes observed.

Pyuria.—Pus in the urine. It results (1) from suppurative inflammation of any part of the genito-urinary tract, and (2) from the rupture of abscesses into the tract.

It appears as a dull, greenish-yellow precipitate that is converted into a clear gelatinous mass by the addition of liquor potassæ. It can always be detected by the microscope.

Source.—If the pus is from the kidney, it is intimately mixed with the urine; the latter usually has an acid or neutral reaction, and the associated symptoms point to the kidneys.

If the pus is from the bladder, it is not so intimately mixed with the urine; the latter is often alkaline in reaction, and the associated symptoms point to the bladder.

Ehrlich's Diazo-reaction.—In certain diseases the urine contains aromatic bodies that produce a characteristic color with sulpho-diazobenzol.

Process.—Two solutions should be prepared and kept in separate bottles:

1. Sulphanilic acid	5.0	2. Sodium nitrite	0.5
Hydrochloric acid, pure	50.0	Distilled water	100.0
Distilled water	1000.0		

In order to apply the test, 50 c.c. of No. 1 are added to 1 c.c. of No. 2. The mixture is added to the urine in a test-tube in the proportion of half urine and half mixture. One c.c. of ammonia-water is then added and the test-tube is violently shaken. The reaction is positive only when the resulting froth acquires a rose-red (not brown)color.

The diazo-reaction is commonly present in typhoid fever. Its value in diagnosis is lessened by its frequent occurrence in tuberculosis, measles, pneumonia, and septic diseases.

TESTS OF THE FUNCTIONAL CAPACITY OF THE KIDNEYS

The most important tests are (1) those which deal with the quantitative determination of the excretion of various substances in the urine, especially urea, sodium chlorid, sugar after phloridzin administration, and certain dyes, particularly phenolsulphonephthalein; and (2) those which deal with the quantitative determination of the concentration of certain substances in the blood, especially urea and incoagulable nitrogen. *Cryoscopy*, or the measurement of the molecular concentration of solutions by ascertaining their freezing points, affords information of some value. In health the freezing point of the blood varies from −0.55° C. to −0.57° C., and that of the urine from −1.30° C. to −2.20° C. In nephritis the freezing point of the blood is lowered and that of the urine is raised, the two figures tending to approximate. A smaller output than intake of *sodium chlorid*, if persistent, is usually indicative of renal inadequacy, and leads to salt retention. The latter bears no direct relation to the degree of nitrogen retention or to the development of uremia, but it is, as a rule, accompanied by edema. The urinary chlorid excretions may be estimated by means of an apparatus constructed on the principle of Esbach's albuminometer. The glycosuria which normally follows the subcutaneous injection of *phloridzin* has been found to be slow in appearing or to be wholly wanting in diseases of the kidneys affecting the renal epithelium. Normally sugar appears in the urine within half an hour and disappears after two to four hours. The results of this test are not entirely reliable in estimating the degree of renal insufficiency. In healthy adults the *total non-protein nitrogen of the blood* ranges between 23 and 29 mg. and the *urea nitrogen* between 12 and 16 mg. per 100 c.c. In chronic nephritis without signs of uremia these values are only moderately increased, but on the approach of uremia there is a marked increase in both the nitrogen and urea concentration. Of the various dyes used to test the excretory capa[illegible] the kidneys, *phenolsulphonephthalein* has been shown to be the most satisfactory. A solution is made containing 6 mg. of the dye to 1 c.c. of salt solution. This is injected into the lumbar muscles under aseptic conditions. Following the injection all the urine excreted is collected at the end of one hour and two hours, is diluted to a suitable amount with alkaline water and its 'phthalein content is estimated by colorimetry. Normally, from 40 to 60 per cent. of the amount injected is recovered in one hour, and from 60 to 80 per cent. in two hours. In severe chronic nephritis the 'phthalein excretion in two hours usually varies from 40 per cent. to 0. In chronic passive congestion (cardiac disease) the elimination of 'phthalein, although often considerably reduced in decompensation, is usually rapidly restored with the re-establishment of compensation. This test in conjunction with the estimation of waste nitrogen in the blood, if repeatedly applied, gives a fairly reliable index of the functional capacity of the kidneys, especially in cases of chronic nephritis. It is also of great value in affording an indication of the relative integrity of the two kidneys in cases of unilateral disease if the urine from each kidney can be obtained by means of the segregator or by catheterization of the ureters.

FLOATING KIDNEY

(Movable Kidney; Nephroptosis)

Definition.—A condition in which the kidney manifests a high degree of mobility.

Etiology.—It is much more frequent in women than in men. Tight lacing, frequent pregnancies, rapid loss of flesh, and over-exertion are reputed causes. Relaxation of the abdominal muscles or mesenteric tissues, congenital or acquired, diminution of the perinephric fat, and preternatural shallowness of the kidney bed are probably the most important etiologic factors.

Symptoms.—The right kidney is the one usually affected, probably from its relation to the kidney, which moves during the respiratory acts. The kidney may be found in any part of the abdomen as a movable tumor, reniform in shape, somewhat tender to the touch, and rarely imparting the pulsation of the renal artery. Not infrequently gastroptosis and enteroptosis coexist.

There may be no subjective symptoms. In many cases, however, there is a sensation of discomfort in the abdomen, accompanied by digestive disturbances, neurasthenic phenomena, a hypochondriasis. Occasionally painful paroxysms occur simulating renal colic (Dietl's crises). These have been attributed by some to engorgement of the kidney from twisting of the renal vessels, and by others to intermittent hydronephrosis.

Diagnosis.—The reniform shape of the tumor, its free mobility, its stationary size, the lessened resistance on percussion over the renal region of the affected side, and the absence of cachexia will serve to diagnose a floating kidney from other abdominal tumors.

Treatment.—In many cases regulation of the diet, the avoidance of undue exertion, the employment of measures intended to increase the body weight and to improve the tone of the abdominal muscles, and the application of a well-fitting supporter will suffice. If the distressing symptoms persist and prove disabling, nephrorrhaphy (stitching the kidney to the posterior abdominal wall) should be considered. Cure or marked relief may be expected to follow operation in about 50 per cent. of the cases.

HYPEREMIA OF THE KIDNEYS

Active Hyperemia.—Acute congestion of the kidneys marks the early stage of acute nephritis and is due to the same causes (exposure to cold, infectious diseases, overdoses of certain drugs, pregnancy, etc.) as the latter. The kidneys are large, soft, and of deep red color. The blood-vessels are engorged and the epithelium shows evidences of cloudy swelling. *Clinically*, the urine is scanty and contains a small amount of albumin, a few blood corpuscles and epithelia, and a few hyaline and finely granular casts. There is no edema or uremia, and the symptoms soon disappear after the removal of the exciting cause. The treatment is that of the milder forms of acute nephritis.

Passive Hyperemia.—This is usually the result of chronic cardiac incompetence, but it may be due to pressure on the renal veins by a tumor, ascites, or pregnant uterus, and occasionally it is caused by thrombosis or embolism of the renal veins or ascending vena cava. The kidneys are enlarged, firm, of a bluish-red color. In long-standing cases the fibrous tissue is increased and the epithelium more or less degenerated (*cyanotic induration*). *Clinically,* the urine is scanty, turbid and of high specific gravity. It contains a small amount of albumin, a few blood corpuscles, and a few hyaline casts. Other evidences of venous stasis (dyspnea, enlargement of the liver, edema, etc.) are usually present. Uremia does not occur unless the condition has resulted in nephritis. The treatment is mainly that of the primary disorder. The patient should be kept at rest. The diet should consist chiefly of milk and farinaceous food. Dry cupping is of service. Digitalis (2 to 4 drams of the infusion) is usually indicated. Hydragogue cathartics (salines or compound jalap powder) are useful adjuvants. In the cardiac cases such a capsule as the following may be of service:

℞.	Pulveris digitalis....................	gr. xx
	Theobrominæ	ʒj
	Extracti nucis vomicæ..............	gr. iv.—M.

Pone in capsulas No. xx.

Sig.—One capsule three or four times a day.

UREMIA

Definition.—The name applied to a group of symptoms resulting from an auto-intoxication conditioned upon impairment of the renal functions. No known constituent of the urine has thus far been identified as the noxious agent.

Symptoms.—Uremia may occur in any type of nephritis or as the result of any condition causing complete suppression of urine. It may be sudden in its onset and rapid in its course (*acute uremia*), or it may develop slowly and persist for weeks or months (*chronic uremia*). In the acute form the most common manifestations are severe headache, intense lessness and jactitation, epileptiform convulsions, de (usually muttering but occasionally maniacal), coma (generally preceded by convulsions, delirium, or severe headache), dyspnea of an asthmatic or a Cheyne-Stokes type, and persistent vomiting, sometimes associated with hiccup and diarrhea. Transient blindness, unaccompanied by any changes in the fundus of the eye, may also occur.

The more chronic manifestations include dull headache, vertigo, ready mental and physical fatigue, recurrent attacks of nausea and vomiting (often regarded as "bilious attacks"), continuous dyspnea, obstinate insomnia, and various mental derangements. Hemiplegia or monoplegia, usually transitory and probably the result of cerebral edema, is not uncommon. Among the less frequent symptoms may be mentioned muscular cramps, twitching of the limbs, tinnitus aurium, itching of the skin and erythematous eruptions.

The tongue is coated, the breath is foul (ammoniacal or urinous), the urine is generally scanty, or if undiminished of low specific gravity, the temperature is, as a rule, subnormal, although it may be elevated (uremic fever), the arterial tension is usually high, but with advanced myocardial degeneration it may be low, the waste nitrogen of the blood is increased and the 'phthalein elimination is decreased and may be nil. The symptoms of chronic uremia may persist with varying intensity for months, but acute exacerbations are likely to occur at any time.

Latent Uremia.—Uremia resulting from complete suppression of urine is marked by insomnia, digestive disturbances, dyspnea, and progressive muscular weakness. Convulsions are absent, and the mind may remain clear almost to the end. Unless the obstruction is removed death supervenes about the tenth or eleventh day.

Diagnosis.—This is based upon the clinical history, the various evidences of nephritis, the state of the arteries and heart, the odor of the breath, and, in doubtful cases, upon the determination of 'phthalein elimination by the kidneys and the urea concentration in the blood. The differentiation of uremic coma from other forms of coma is considered on page 445.

Prognosis.—This is always grave. Recovery is possible, however, even after the most severe symptoms.

Treatment.—The chief indication is to favor elimination. One or two drops of croton oil, diluted with glycerin, or elaterium (1/6–1/4 gr.) should be given at once. Sweating should be promoted by hot-air or vapor baths. Occasionally the subcutaneous administration of pilocarpin (1/6–1/4 gr.) may be desirable, but it should be employed with caution. In robust subjects, especially if coma or convulsions appear, venesection may be practised, the removal of from 10 to 20 ounces of blood often proving very efficacious. In children a few ounces of blood may be abstracted from the loins by means of wet cups. After the blood has been withdrawn an equal volume of normal saline solution may be given intravenously. Irrigation of the bowel with hot saline solution (105° F.) is also useful. Not rarely lumbar puncture is of value. Convulsions may be controlled by a few whiffs of chloroform, by an enema of chloral (20 to 30 grains), or by injection of morphin (1/4 gr.).

Chronic uremia must also be treated by measures intended to lessen the concentration of toxic matters in the blood. The most important of these measures are restrictions in diet, rest, free purgation (salines or compound jalap powder), and copious sweating by means of hot-air or vapor baths. Digitalis, strophanthus, theobromin, etc., are often of service even when arterial tension is high. Morphin is especially useful in controlling asthmatic seizures, restlessness and insomnia.

ACUTE NEPHRITIS

(Acute Bright's Disease; Acute Diffuse Nephritis; Acute Parenchymatous Nephritis; Acute Catarrhal Nephritis)

Definition.—An acute inflammatory disease of the kidney, involving especially the epithelium of the tubules and glomeruli.

Etiology.—The chief causes are: (1) Infectious diseases, especially scarlet fever; (2) poisons which are eliminated through the kidneys, such as cantharides, turpentine, etc.; (3) exposure to cold and wet; and (4) pregnancy. Less frequently it occurs in connection with acute tonsillitis, acute gastro-enteritis, purpuric affections, inflammatory skin diseases or extensive burns.

Pathology.—The kidney is swollen and the capsule non-adherent. At first the organ is bright red in color; it soon, however, becomes pale and mottled in appearance, although the Malpighian tufts still retain their deep red tint.

Histology.—The epithelium of the tubules and glomeruli is the seat of cloudy swelling and, later, of fatty degeneration. Desquamated epithelium, blood-corpuscles, and an albuminous exudate block the tubules. The capillaries are dilated, their walls are degenerated, and bloody extravasations are not infrequently seen. The interstitial tissue is more or less infiltrated with leukocytes.

Symptoms.—In many cases the only indications of acute nephritis are the urinary changes with perhaps, the occurrence of dropsy. In the more severe cases the general symptoms are slight fever and its associated phenomena; dull lumbar pain; nausea and vomiting; dropsy, beginning in the face and becoming general; and pronounced anemia. Increased arterial tension and accentuation of the aortic second sound are often observed. Uremic symptoms may develop at any time. (see p. 139).

The Urine.—The urine is scanty and at times suppressed. It is smoky in appearance, of high specific gravity, and contains a large amount of albumin, free blood, epithelial cells and hyaline, blood, epithelial and granular casts. The total output of urea is decreased, although the percentage may be increased.

Complications.—The most important are secondary infections, bronchopneumonia, inflammation of the serous membranes (pleuritis, pericarditis), edema of the lungs, edema of the larynx, and dilatation of the heart.

Diagnosis.—As the general symptoms are often slight, the diagnosis must rest on the examination of the urine. The history and the absence in the urine of wide, highly fatty casts will serve to distinguish *acute nephritis* from an *acute exacerbation of chronic parenchymatous nephritis.*

Prognosis.—This is uncertain, although in general it is favorable, recovery ensuing in the majority of cases in from a few weeks to several months. A fatal issue may be the result of exhaustion, uremia, or one of the complications already mentioned. In some instances the disease becomes chronic.

Treatment.—Absolute rest in bed for from four to six weeks is imperative. Milk largely diluted with carbonated water, Vichy, or lime-water is the best food. Beef-tea and broths should be interdicted.

In the absence of any direct remedies the indications are to divert the blood from the inflamed kidneys, to lessen their work as much as possible by increasing the action of the bowels and skin, and to meet the symptoms as they arise.

At the onset, if there be pain or suppression of urine, dry cupping, or, in severe cases, wet cupping over the region of the kidney is of value. Following the cupping warm poultices may be applied to the loins with advantage. Cantharides, turpentine, or similar drugs should not be used.

The bowels should be kept freely opened by means of daily purges, the best being salines in concentrated solution and compound jalap powder (20 to 30 grains).

Free sweating is very useful in promoting elimination by the skin. It may be effected by means of hot-water baths, hot packs, vapor baths, hot-air baths, or the subcutaneous administration of pilocarpin ($\frac{1}{10}$ to $\frac{1}{6}$ grain).

Unirritating diuretics, like digitalis and potassium citrate or acetate, are useful. These drugs may be combined, as in the following formula:

℞. Potassii acetatis.................... ʒij
Infusi digitalis...................... f℥iij.—M.
SIG.—A tablespoonful, well diluted, thrice daily.

Excessive *dropsy* may demand puncture of the swollen parts, a free incision at the outer side of each ankle, or the insertion of delicate silver cannulæ (Southey's tubes) beneath the skin. Uremia will call for its appropriate treatment (see p. 141). After the acute symptoms have subsided, iron may be employed in the form of Basham's mixture (2 to 4 fluidrams) to combat anemia.

CHRONIC PARENCHYMATOUS NEPHRITIS

(The Large White Kidney and Contracted White Kidney)

Etiology.—It may follow acute nephritis or it may be chronic from the beginning. Habitual exposure, abuse of alcohol, chronic infections (tuberculosis, malaria), and passive congestion from heart disease are predisposing factors. It occurs most often between the ages of twenty and forty.

Pathology.—The kidney may be large or small. In the large type the organ is soft and of a grayish-yellow color (*large white kidney*) or mottled with reddish and yellow areas (*large red or variegated kidney*). The capsule is thin and strips readily. The cortex is swollen and of an opaque yellowish-white color, with punctate hemorrhages. Microscopically, there is marked fatty degeneration of the tubular and glomerular epithelium. Many tubules and capsular spaces are filled with epithelial cells in various stages of degeneration. The walls of the capillaries may show hyaline changes. Small hemorrhages are frequently seen, the color of the organ being red rather than yellow (fatty degeneration) if these are numerous and extensive. The intertubular connective tissue is somewhat edematous and slightly increased.

In the second form (the more common type) the organ is small, pale and firm (*contracted white kidney*). The surface is uneven and the capsule is thickened and in places adherent. Microscopically, the lesions are much the same as in the large white kidney, except that the fibroid changes are extensive and advanced. Whether the contracted white kidney is a terminal stage of the large white kidney or is a distinct disease is a mooted question. Forms intermediate between the two are often observed and many others which cannot be sharply differentiated from chronic interstitial nephritis (*mixed forms*).

Symptoms.—The symptoms usually develop insidiously and consist of progressive weakness, marked anemia, general dropsy (often first noted in the face on rising in the morning), digestive disturbances, and sooner or later a variable degree of cardiac hypertrophy with increased arterial tension and accentuation of the aortic second sound, these cardiovascular features being especially pronounced with the contracted white kidney. Uremic symptoms may develop at any time.

The Urine.—In the large white kidney the urine is lessened in amount, usually turbid, of increased specific gravity (1018–1025), highly albuminous, deficient in urea, and microscopically shows numerous fatty epithelial cells, blood-corpuscles, and casts of nearly every variety, broad casts containing coarse dark granules or oil globules and waxy casts being especially abundant. In the contracted white kidney the urine is increased in amount, of low specific gravity, and contains fewer casts and a smaller percentage of albumin than in the large white kidney.

Complications.—These are numerous and often suggest the diagnosis. The most common are uremia, extensive serous effusion into the tissues or serous cavities, latent inflammation of the serous membranes, pneumonia, dilatation of the heart, albuminuric retinitis, apoplexy, and acute exacerbations.

Prognosis.—The outlook for cure is unfavorable. The duration is commonly from a few months to several years.

Treatment.—The treatment is largely dietetic and hygienic. Residence in a dry, warm, and equable climate serves to prolong life. Rest is an essential element in the treatment. The underclothing should be woolen or silk. The diet should be relatively non-nitrogenous but of sufficient caloric value, and in severe cases an exclusive milk and farinaceous diet, at least for a time, may be absolutely necessary. Warm baths with friction are useful in promoting free action of the skin, but great care must be exercised after their use to avoid chilling. The bowels should be kept active by natural mineral waters or saline laxatives. If the urine is scanty, digitalis, caffein, potassium citrate, or theobromin may prove efficacious. The following combination sometimes acts beneficially:

℞.	Theobrominæ	gr. xxxvj
	Caffeinæ citratæ	gr. xii
	Lithii benzoatis	ʒj.—M.

Fiant chartulæ No. xij.
Sig.—One powder four times a day.

Basham's mixture is often useful as a hematinic, but only small doses should be used. Strychnin and the simple bitters are valuable adjuvants to iron in many cases. Excessive dropsy will call for a comparatively dry diet with as little salt as possible, for hydragogue cathartics (Epsom salts, compound jalap powder, or elaterium), for diaphoretics (hot-air baths), and perhaps for operative measures (puncture of the legs and scrotum, insertion of Southey's tubes, incisions near the ankles, aspiration of serous sacs). Uremia will demand special treatment (see p. 141); acute exacerbations should be treated as primary attacks of acute nephritis.

CHRONIC INTERSTITIAL NEPHRITIS

(Red Granular Kidney; Contracted Kidney; Gouty Kidney)

Definition.—A chronic disease of the kidney characterized by a marked overgrowth of its connective-tissue elements, and almost invariably associated with general arteriosclerosis and hypertrophy of the heart.

Etiology.—It is much more common in males than in females, and is most frequently encountered between the ages of forty and sixty. (1) In many cases it can be traced to an irregular mode of life (overeating, insufficient muscular exercise, nervous strain, etc.), gout, chronic alcoholism, lead poisoning, syphilis or other infectious diseases; (2) it is a common accompaniment of arteriosclerosis; (3) it may follow passive congestion, as from chronic heart disease.

Pathology.—The kidney is small and red in color. The surface is granular and the capsule adherent. The organ is firm, cuts with difficulty, and on section often reveals small cysts or calcareous deposits. The cortical substance is greatly reduced in thickness. Microscopic examination shows an overgrowth of fibrous connective tissue, which is especially marked around the glomeruli and small arteries, and more or less atrophy of the parenchyma. The blood-vessels show mural thickening. Many of the glomeruli are completely destroyed or have undergone hyaline degeneration, and many of the tubules have collapsed or have dilated to form minute cysts.

The arteries throughout the body are the seat of sclerotic changes, in consequence of which hypertrophy of the heart, especially of the left ventricle, has resulted.

Symptoms.—The symptoms develop most insidiously. There is gradual loss of strength, with increasing anemia. Gastric disturbances are common. Vascular symptoms are prominent, and include thickening of the arterial walls, high arterial tension, accentuation of the aortic second sound, and hypertrophy of the heart. Dyspnea is present in the late stages, and may result from cardiac weakness, edema of the lungs, hydrothorax, or uremia. Headache, vertigo, and insomnia often result from the disturbed circulation or from uremia. Dimness of vision from albuminuric retinitis is a serious symptom. Dropsy is often absent, or is slight and late in appearing. Uremia is of very frequent occurrence.

The Urine.—The urine is copious (80 to 150 ounces), pale in color, of low specific gravity—1005 to 1012—and contains but a trace of albumin and few narrow hyaline or pale granular casts. Hematuria is sometimes noted.

Complications.—These include albuminuric retinitis, dilatation of the heart, cerebral hemorrhage resulting from the weakened arteries and large heart, uremia, inflammation of serous membranes (often latent), acute pulmonary edema, pneumonia, and bronchitis. Nose-bleed and other hemorrhages are not uncommon.

Diagnosis.—*Chronic parenchymatous nephritis* usually occurs in younger subjects and runs a shorter course. There is decided edema, and the urine is usually decreased in quantity and contains much albumin and wide fatty casts.

Prognosis.—The disease is incurable, but may last many years. The possibility of acute uremia, cerebral hemorrhage, or secondary infection must be borne in mind.

Treatment.—The dietetic and hygienic treatment is that of chronic parenchymatous nephritis. Frequent tepid baths with friction of the skin are advantageous. The bowels should be kept regular with mild saline cathartics or alkaline mineral waters. Nitroglycerin ($\frac{1}{150}$ to $\frac{1}{50}$ grain) is often useful when arterial tension becomes excessive and causes headache, vertigo, palpitation, and dyspnea. Basham's mixture in small doses (1 fluidram) is sometimes of service if there is pronounced anemia. When there is severe insomnia, bromids, chloral, paraldehyd, and trional may be tried in the order named. Morphin is sometimes indispensable. Heart-failure with low arterial tension will require the use of such stimulants as digitalis, strychnin, caffein, and theobromin.

AMYLOID DEGENERATION OF THE KIDNEY

(Waxy Kidney; Lardaceous Kidney)

Etiology.—It occurs in prolonged suppurative diseases, especially of bones, in tuberculosis, syphilis, and cachectic states.

Pathology.—The kidney is enlarged, firm, and pale, and on section may present a waxy, translucent appearance. The amyloid areas are colored mahogany brown by the application of Lugol's solution to the cut surface. Other organs, especially the liver and spleen, usually share in the degenerative process.

On microscopic examination the walls of the blood-vessels, especially of those of the Malpighian bodies, are found thickened and infiltrated with a homogeneous wax-like material that turns pink when treated with gentian-violet. The epithelium is often fatty.

Symptoms.—Most patients appear badly nourished and anemic. Dropsy is present in many cases. The liver and spleen are usually enlarged from the same cause. Uremia is very rare.

The Urine.—The urine is increased in quantity, is rich in albumin, and is of low specific gravity. Microscopically, it contains hyaline and waxy tube casts and degenerated epithelium.

Diagnosis.—This is based upon the history, the enlargement of the liver and spleen, and the polyuria with marked albuminuria.

Prognosis.—In the majority of cases the prognosis is very grave. In the early stages an arrest of the process is not impossible if the original disease can be cured.

Treatment.—The treatment is chiefly that of the primary disease. In other respects it must be purely hygienic, dietetic, and symptomatic.

PYELITIS AND PYELONEPHRITIS

Inflammation of the pelvis of the kidney (*pyelitis*) and inflammation of the kidney substance frequently coexist, one condition causing the other. The conjoint affection, which is known as *pyelonephritis*, is often suppurative. Pyelitis may be catarrhal, suppurative or ulcerative; unilateral or bilateral; and acute or chronic.

Etiology.—Pyelitis or pyelonephritis is always the result of infection reaching the kidney or its pelvis from the lower urinary tract (urogenous infection), from the blood stream (hematogenous infection), or directly by contiguity from some adjacent structure (colon, appendix, etc.), or by means of wound. The bacteria most frequently concerned in tl process are the colon bacillus, the pus cocci, the typhoid bacillus, and the Bacillus proteus vulgaris. Among the predisposing causes of infection may be mentioned general infections, trauma, exposure to cold, gastro-intestinal diseases, excretion of irritating drugs, calculi in the bladder or renal pelvis, cystitis, and especially obstruction of the urinary passages with stasis of urine (urethral stricture, prostatic hypertrophy, pelvic tumors, pregnancy, etc.).

Pathology.—The kidney is pale, more or less enlarged and edematous. In the obstructive cases the pelvis is distended at the expense of the kidney substance. The lining membrane is injected, covered with mucopus, and not rarely ulcerated. In suppurative pyelonephritis the renal substance may be riddled with miliary abscesses or may be almost completely destroyed, the whole organ being transformed into a large multilocular pus sac (*pyonephrosis*). In long-standing cases the pus may undergo inspissation and calcification, forming putty-like or mortar-like masses.

Symptoms.—The symptoms are often vague and overshadowed by those of the primary disease. Constitutional disturbances, varying markedly in degree, are usually present in the acute cases and in exacerbations of chronic cases, the most common being chills, fever (100° to 104° or more), sweats, leukocytosis, digestive disturbances, and loss of weight and strength. In addition there may be local pain, tenderness in the kidney region, frequent micturition, and in the event of pyonephrosis a smooth rounded tumor. The urine contains pus, mucus, epithelial cells, blood cells, bacteria, albumin, and in pyelonephritis casts of various forms. A larger amount of albumin than can be accounted for by the pus and blood suggests involvement of the kidney. The amount of urine varies. In acute cases it is usually diminished and in chronic cases, unless both kidneys are seriously affected, it is generally increased. In acute cases the reaction of the urine is usually acid, but in the chronic forms it is often neutral or alkaline.

Diagnosis.—An exact diagnosis can almost always be made from the history of the case, a thorough physical examination, careful urinalysis, including cultures (catheterized specimens), functional tests (see p. 136), cystoscopy, ureteral catheterization and radiography. In *cystitis* pain is referred to the hypogastric region, there is pronounced dysuria, the urine is more likely to be alkaline in reaction than acid, and lumbar symptoms and signs are absent.

Calculous Pyelitis.—Sharp pain, increased by jarring movements, and reflected down the ureters, and the presence of much blood in the urine point to calculous pyelitis. Ureteral catheterization and *x*-rays are valuable aids in diagnosis.

Renal tuberculosis may be recognized by the history, the presence of tuberculous foci in other organs, and the discovery of tubercle bacilli in the urine. The tuberculin test may also aid in the diagnosis.

Prognosis.—Mild acute attacks of pyelitis usually end in recovery. In acute pyelonephritis much depends upon the character of the infection, the extent of the renal involvement, and whether the disease is unilateral or bilateral. Chronic pyelitis and chronic pyelonephritis may last for years. The most grave cases of the latter are those in which both kidneys are involved as a result of obstruction and in which the urinary reaction is alkaline.

Treatment.—In acute cases the important measures are removal of the cause, if possible, rest in bed, the application of cups or warm compresses over the kidney region, a simple unirritating diet, the liberal use of diluents, free catharsis, preferably by means of copious warm enemas, and the administration of antiseptics, such as hexamethylenamin (5–10 grains three or four times a day), and, if necessary, remedies to modify the reaction of the urine, such as potassium citrate in one case and benzoic acid in another. If pain is severe morphin will usually be required. In protracted cases autogenous vaccines are sometimes useful. In chronic pyelonephritis special attention must be paid to the underlying condition (stricture, prostatic hypertrophy, cystitis, calculus, or a contiguous focus of infection). Surgical intervention is often required.

NEPHROLITHIASIS

(Renal Calculus; Gravel)

Definition.—Renal calculi are concretions formed in the kidney by the precipitation of various solid constituents of the urine.

Etiology.—The disease is more common in males than in females. Heredity and sedentary habits are given as predisposing causes. The formation of stones is favored by the presence in the urine of any sparingly soluble substance in excess. Mucus, blood, pus, or epithelium may furnish the nucleus.

Pathology.—The size of renal concretions varies from that of coarse sand ("gravel") to that of a large bean. Several stones are often present and in from 15 to 20 per cent. of the cases both kidneys are affected. The most common forms are those composed of—(1) Uric acid and its compounds; (2) oxalate of calcium; (3) phosphate of calcium and of ammonio-magnesium phosphate. Stones composed of xanthin and cystin are rare.

Uric acid stones are the most common. They are usually smooth, of a reddish-brown color, and comparatively hard. *Oxalate calculi* are very hard, of a dark brown color, and uneven (mulberry calculi). *Phosphatic calculi* are grayish-white in color, soft, and mortar-like.

Events.—(1) Small particles are frequently passed without serious disturbance. (2) Larger concretions may be extruded with intense pain (*renal colic*). (3) Calculi may remain in the pelvis and excite pyelitis or pyelonephritis. (4) They may obstruct the ureter and cause hydronephrosis or pyonephrosis, or if the obstruction is complete, atrophy of the kidney.

Symptoms.—*Pain and tenderness* in the kidney region are common symptoms. The pain is aggravated by rough motion, and tends to radiate along the ureter. *Irritability of the bladder* is sometimes a prominent feature. The urine frequently contains *blood*, *pus*, *epithelium*, and *crystals* indicating the nature of the stone.

Symptoms of Sepsis.—Irregular fever, chills, sweats, leukocytosis, and pallor often mark the occurrence of suppurative pyelitis. *Renal colic* is excited by the entrance of the stone into the ureter. It is characterized by intense pain radiating from the kidney downward into the groin, thigh, and testicle. The testicle is often retracted. There are often nausea, vomiting, and collapse. After such an attack the urine may contain blood or particles of stone. *Anuria* is one of the most serious complications.

Diagnosis.—Radiography, cystoscopy, and ureteral catheterization are valuable aids to diagnosis. In the differential diagnosis one must exclude biliary colic, intestinal colic, Dietl's crises, tumor of the kidney, tuberculosis of the kidney, prostatic disease (hypertrophy, cancer, chronic prostatitis), vesical calculus, disease of the spine, and lumbago.

Prognosis.—In uncomplicated cases the prognosis is good under appropriate treatment.

Treatment.—This should be directed to the underlying diathesis. In cases of uric-acid calculi alkalis and alkaline mineral waters are useful. A quart of water containing 40 grains of potassium bicarbonate and 20 grains of lithium citrate may be taken daily. Special remedies, like piperazin, lycetol, and urosin, have been recommended as solvents, but they are of doubtful value. If phosphatic calculi are present, benzoic or boric acid (5 to 15 grains thrice daily) may be employed in a similar manner.

Operation (nephrolithotomy, pyelotomy, or nephrectomy) is urgently demanded if the attacks of renal colic occur with such frequency as to prove disabling, if there are evidences of severe pyelitis, or if there is reason to believe that the calculus has become permanently impacted in the ureter.

Renal Colic.—The indications are to relieve the pain ar to relax the spasm. This is best accomplished by hypodermic injections of morphin and atropin, coupled with hot baths or local applications—hot poultices or fomentations. If the pain is extreme, it may be desirable to administer chloroform. Simple diluents should be given freely. In mild attacks full doses of phenacetin or antipyrin, with an abundant supply of hot drinks, may suffice.

HYDRONEPHROSIS

Definition.—Overdistention of the renal pelvis with urinous fluid, the result of mechanical obstruction.

Etiology.—The chief causes are: (1) Congenital stricture, twisting or other anomaly of the ureter. (2) Impaction of a calculus in the ureter. (3) Abdominal tumors compressing the ureter. (4) Tumors growing within the urinary passages. (5) An inflammatory stricture of the ureter or urethra. (6) Compression of the urethra by an enlarged prostate.

Pathology.—The pelvis reveals all grades of distention. In extreme cases it may contain several quarts of clear or turbid fluid, of low specific gravity, and exhibiting traces of the normal urinary ingredients. There is more or less atrophy of the renal tissue. Both kidneys are usually affected when the obstruction is in the lower urinary tract.

Symptoms.—Slight distention yields no symptoms. In other cases a tumor slowly develops in the region of the affected kidney. Not rarely the tumor periodically disappears and reappears, the disappearance being associated with the passage of a large amount of urine (intermittent hydronephrosis). Large cysts are often painful.

Diagnosis.—This should be based upon the history and the exclusion of other abdominal enlargements.

Pyonephrosis may usually be excluded by the absence of purulent urine and symptoms of sepsis.

Ovarian cysts enlarge upward from the pelvis, are more mobile, more superficial, less likely to be overlaid by intestine, and more likely to displace the uterus.

Prognosis.—If the cyst is unilateral and of no great size, life may be indefinitely prolonged. The disease may end fatally in consequence of rupture into the peritoneum, secondary pyonephrosis, or, if bilateral, of uremia.

Treatment.—Large accumulations will demand surgical treatment; small ones should not be disturbed.

TUBERCULOSIS OF THE KIDNEY

Etiology.—The etiology of renal tuberculosis is that of tuberculosis in general. The infection may be hemogenic, lymphogenic, or urogenic. Males are more frequently attacked than females. The majority of cases are encountered between the ages of twenty and forty years.

Pathology.—Two forms of renal tuberculosis have been recognized—the miliary and the caseous. The former is nearly always bilateral, is an acute process, and is generally unmistakably secondary to tuberculosis elsewhere in the body. The caseous variety runs a chronic course; it usually begins as a unilateral affection, although the other organ is commonly ultimately involved, and a primary focus may or may not be apparent in some other structure. The ureter and bladder are often affected, and in this event either the kidney or the bladder may have been the starting point of the infection in the urinary tract.

Symptoms.—The chief symptoms are: pain in the lumbar region, usually dull, but sometimes sharp like that of renal colic; tenderness on pressure; frequency of urination; dysuria; slight, irregular fever, and more or less cachexia. The urine is usually acid in reaction, and may contain pus, blood, albumin, tubercle bacilli, cheesy particles, and débris. Tube casts are rarely found. In many cases some enlargement of the affected organ can be detected by bimanual palpation.

Diagnosis.—*Calculous Pyelitis.*—In this condition pain is usually more severe and more likely to be affected by movement. Hematuria is more profuse, and is often excited by exertion. Cachexia is not so marked, and there are no tubercle bacilli in the urine. The tuberculin test, x-rays, or catheterization of the ureter may decide the diagnosis.

Tumors of the kidney grow rapidly and usually reach a very large size, produce copious hematuria, but rarely cause pyuria, and are often painless.

Prognosis.—This is always grave. Without intervention the duration is from a few months to several years.

Treatment.—When the renal disease appears to be primary and unilateral, and the patient's strength will permit, nephrectomy should be recommended. The operative mortality is from 3 to 10 per cent. Complete cure is the rule in the early stages. In advanced cases the treatment must, of necessity, be palliative.

POLYCYSTIC DISEASE AND MALIGNANT TUMORS OF THE KIDNEY

Polycystic Disease.—In this condition, which is usually congenital but not rarely latent until adult life, the kidney is greatly enlarged and composed of a vast number of cysts varying in size from that of a pea to that of a walnut. The disease is generally bilateral and probably depends upon a defect of development. Clinically it is characterized by the occurrence of tumor masses in both renal regions, intermittent hematuria, and the urinary and cardiovascular phenomena of chronic interstitial nephritis. Operation is only justifiable in the rare unilateral cases.

Malignant Tumors.—The primary malignant growths of the kidney comprise sarcoma, hypernephroma, and carcinoma. Sarcoma is the most common tumor in children and hypernephroma is the most common tumor in adults. Carcinoma is comparatively rare. The chief symptoms are progressive emaciation, cachexia, hematuria (often profuse), and the occurrence of a tumor springing from the lumbar region, overlaid by the colon, sometimes movable in respiration and by palpation, and usually separated from the liver or spleen above by an area of resonance. Pain is inconstant and the urine, apart from the hematuria, often affords no indications. Metastasis is frequently observed, hypernephromas showing a special tendency to involve the lungs and bones and to invade the renal vein and vena cava. Except for intolerable pain or uncontrollable bleeding, operation is contraindicated if metastases are found.

DISEASES OF THE BLOOD AND THE BLOOD-FORMING ORGANS

NORMAL BLOOD

In health the blood amounts to about one-thirteenth of the body-weight. Normally there are approximately 5,000,000 red blood-corpuscles in the cubic millimeter. This number is temporarily diminished during menstruation, gestation, lactation, and fatigue, and after the ingestion of much fluid. Fasting and profuse sweating increase the number of red cells by concentrating the blood. In the first few days of life the number in a cubic millimeter may be 7,000,000 to 8,000,000. In high altitudes the number is also increased. There are from 5000 to 10,000 white cells in the cubic millimeter, the ratio of white to red cells being about 1 to 500. The number of blood-plates is from 200,000 to 700,000.

EXAMINATION OF THE BLOOD

A clinical study of the blood has for its object the determination of the percentage of hemoglobin, the specific gravity, the alkalinity, the number, form, and relative proportion of the various corpuscles, and the detection of free pigment, bacteria, and animal parasites.

Estimation of Hemoglobin.—The percentage of hemoglobin may be determined by either Fleischl's or Tallqvist's hemoglobinometer, although the former is preferable.

Fleischl's instrument consists of a metal stand with a circular aperture in the center, under which is placed a reflector made of plaster-of-Paris. The aperture is fitted with a small cell having a glass bottom, and divided into two equal compartments. A graduated wedge of colored glass is employed as a standard, the 100 on the scale being intended to represent the percentage of hemoglobin in normal blood. This wedge of glass is so arranged that when moved under the stand, one compartment of the cell will receive white light from the reflector, and the other, red light from the tinted glass. A small capillary tube is held over a drop of blood until filled, and is then washed in one of the compartments of the cell, in which has been previously placed some distilled water. Both compartments are then equally filled with water, and the wedge of glass is moved by means of a thumb-screw until the tints in the two chambers are exactly the same, when the percentage of hemoglobin may be read off.

In the examination it is necessary to use artificial light. The 100 mark on the scale, which is intended to represent the percentage of hemoglobin in normal blood, is too high for the average person, 85 or 90 per cent. rarely being exceeded.

In *Tallqvist's method* the color of a drop of undiluted blood soaked into a standard filter paper is compared with tints ranging from 10 per cent. to 100 per cent. on a prepared color scale. This method is very simple and sufficiently accurate for practical purposes.

The Specific Gravity of the Blood.—The specific gravity of the blood in health varies from 1050 to 1070. In grave anemia it is often considerably diminished. Hammerschlag's method consists in expelling a drop of blood into a mixture of chloroform and benzol, one or the other of these substances being subsequently added until the drop neither rises nor falls. The specific gravity of the mixture may then be ascertained in the usual way. Lloyd Jones employs mixtures of glycerin and water of different densities, and notes the specific gravity of the mixture in which the blood-drop remains stationary.

Alkalinity of the Blood.—The alkalinity of the blood may be determined by titrating with a standard solution of acetic acid until a change of color is produced when a drop is placed on a plaster-of-Paris plate impregnated with neutral litmus.

Enumeration of Red Blood-corpuscles.—The best instrument for blood counting is the hemocytometer of Thoma-Zeiss. This consists of a glass slide in the center of which is a cell $\frac{1}{10}$ mm. in depth. The floor of the cell is divided into 400 small squares, each of which has an area of $\frac{1}{400}$ square millimeter. These small squares are grouped into sets of 16 by a series of additional vertical and horizontal lines bisecting each fifth column of squares. As the depth of the cell from the cover-glass is $\frac{1}{10}$ mm., the cubic contents of each small square is $\frac{1}{4000}$ cm.

The blood is mixed in a *mélangeur*—that is, a capillary tube one extremity of which is blown into a bulb having a capacity of 100 cm. The mélangeur is marked at 0.5, 1, and 101. A drop of blood issuing from a prick of the finger is sucked cautiously into the tube exactly to the mark 0.5. The point of the tube is quickly wiped dry and immersed in the diluting fluid (2.5 per cent. solution of potassium bichromate or Toison's fluid),[1] which is drawn up to the 101 mark. The instrument is now shaken to secure diffusion of the blood. The diluting fluid remaining in the stem of the mélangeur is now blown out, and a drop of the mixture placed upon the middle of the bottom of the divided cell. The drop in the cell should be free from bubbles, and the cover-glass so adjusted that concentric rings of color appear at the points of contact between the cover-glass and the glass plate. Before counting, a few minutes should be allowed for the corpuscles to settle to the bottom of the cell. The number of corpuscles is then counted in 400 small squares. To avoid repetition in counting, corpuscles on the upper and left boundary-lines should be counted, but those on the lower and right boundary-lines should be disregarded. The number of corpuscles in each cubic millimeter of blood is determined by multiplying the number of corpuscles counted by the degree of dilution (200) and again by the cubic contents of each square (4000), and then dividing the product by the number of squares counted (400). Thus, if 2000 corpuscles were counted in 400 squares, the number of corpuscles in each cubic millimeter would be 4,000,000:

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[1] Methyl-violet, 5 B, 0.025 gm.; sodium chlorid, 1 gm.; pure sodiu sulphate, 8 gm.; neutral glycerin, 30 c.c.; distilled water, 160 c.c.

$$\frac{2000 \times 200 \times 4000}{400} = 4,000,000$$

After using, the mélangeur should be carefully washed in water, alcohol, and ether.

Enumeration of White Blood-corpuscles.—For counting the white blood-cells a mélangeur should be used which allows a dilution in the proportion of 1:10 and an aqueous 0.5 per cent. solution of acetic acid, to which may be added a little methyl-violet, should be selected as a diluting fluid. The red cells disappear in this solution, and the white cells remain and are readily counted. The latter should be counted in 800 small squares. The number of leukocytes in each cubic millimeter is then determined by multiplying the whole number counted by 4000, and again by 10, and dividing by 800.

The Study of the White Blood-corpuscles.—In normal blood the following forms of leukocytes may be recognized.

1. *Small Lymphocytes.*—These are small cells about the size of the red blood-corpuscles. The nucleus is very large and spheric, and stains intensely with basic stains (methylene-blue). With Ehrlich's triacid mixture the nucleus is pale. The narrow rim of protoplasm surrounding the nucleus is non-granular (hyaline). Small lymphocytes constitute from 25 to 35 per cent. of all leukocytes.

2. *Large Lymphocytes.*—These cells resemble those just described, but are considerably larger. The nucleus is relatively not so large, and stains less deeply. In some forms the nucleus is more or less bent or indented (transitional leukocytes). Normally, large lymphocytes make up from 5 to 10 per cent. of the blood-corpuscles.

Polymorphonuclear Neutrophiles.—These cells are somewhat smaller than large lymphocytes, and are actively ameboid. The nucleus appears to be divided into two or more segments and stains deeply. The protoplasm is studded with fine granules, which do not stain well with either simple basic stains (methylene-blue) or simple acid stains (eosin). With Ehrlich's triacid mixture the granules are colored violet and the protoplasm pale pink. Neutrophiles make up from 60 to 70 per cent. of the white cells of normal blood.

Eosinophiles.—These resemble the polymorphonuclear neutrophiles, but are more irregular in outline, and the granules are larger, more highly refractive, more loosely attached, and have a special affinity for acid stains (eosin). Eosinophiles make up from 1 to 4 per cent. of the leukocytes.

Mast-cells (*Basophiles*).—These cells have a lobulated nucleus. The protoplasm is studded with granules having an intensely basic reaction. These granules remain unstained with Ehrlich's triacid mixture, but with methylene-blue they stain deep blue. Mast-cells are only occasionally encountered in normal blood.

In disease, additional forms are sometimes found. Thus, in leukemia large cells are found which are non-ameboid and which have a single round or oval nucleus imbedded in protoplasm containing neutrophilic granules. These have been termed *myelocytes.*

With the aid of a $\frac{1}{12}$-inch oil-immersion lens large and small leukocytes can readily be distinguished in preparations of fresh blood, but to study satisfactorily the various forms it is necessary to dry and then stain the specimen.

The Drying and Staining of Blood.—A small drop of blood, secured by pricking the finger, is spread into a film by being pressed between two perfectly clean cover-glasses, which are then drawn apart and exposed to the air until dry. The cover-glasses should be handled with forceps, since the moisture of the fingers distorts the corpuscles. The preparation is first "fixed" by heating on a copper bar for from one-half to one hour at a temperature of from 100° to 110° C., or by immersing for from five to fifteen minutes in a mixture of equal parts of absolute alcohol and ether.

A convenient method of staining is the one suggested by Stengel. The fixed preparation is immersed for a few minutes in a 1 per cent. solution of eosin in 60 per cent. alcohol, to which has been added an equal quantity of water at the time of staining. The cover-glass is then washed in water and counterstained in Delafield's hematoxylin for a minute, and finally washed, dried, and mounted. The eosinophile granules are dark red, the red corpuscles lighter red, and the nuclei of the leukocytes almost black. Ehrlich's triacid mixture (methyl-green, orange G, and acid-fuchsin) makes an excellent stain. The film should be fixed by heat and flooded with the stain. After the lapse of from five to eight minutes the stain should be washed off in running water and the film dried by gentle heat and mounted in xylol balsam. With this stain the red cells are tinged orange, the nuclei of the leukocytes greenish blue, the neutrophile granules violet, the eosinophile granules red, the nuclei of normoblasts purple, and the nuclei of macroblasts greenish blue.

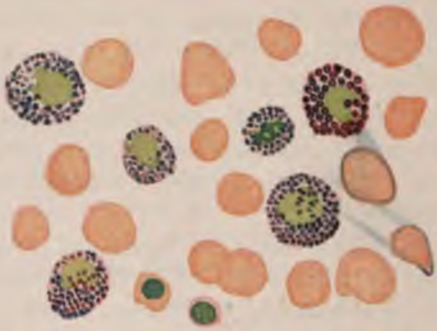

FIG. 6.—Blood in lienomedullary leukemia, showing several mononuclear neutrophiles (myelocytes), one polymorphonuclear neutrophile, and an eosinophile; a nucleated red corpuscle and a lymphocyte are seen in the lower part of the illustration. Stained with Ehrlich's triple mixture (from Stengel's *Text-book of Pathology*).

PLETHORA

An increase in the whole quantity of blood. It is very doubtful whether such a condition can be more than transitory.

HYDREMIA

An excess of water in the blood. As a loss of corpuscular elements is generally replaced by the addition of water extracted from the tissues, most anemias are associated with hydremia. The condition is more marked in general dropsy. Temporary hydremia is produced by the excessive ingestion of fluids.

ANHYDREMIA

A deficiency of fluid in the blood. It is observed in starvation, immediately after hemorrhage, and after copious discharges. as in cholera.

MELANEMIA

A condition in which free pigment granules occur in the blood. It is met with in malaria and certain other fevers, and occasionally in melanosarcoma and in Addison's disease. The pigment may be found in the plasma or in the leukocytes.

POLYCYTHEMIA

Polycythemia, or an increase in the number of red cells, is an apparent condition in blood taken from cyanosed parts. It is observed temporarily in the new-born, in recovery from certain anemias, after transfusion of blood, and in blood concentrated by excessive discharges. Marked polycythemia is sometimes produced by residence in high altitudes and by certain poisons, such as phosphorus and carbon monoxid. It also occurs in the condition known as chronic splenomegalic polycythemia.

MICROCYTOSIS AND MACROCYTOSIS

Microcytosis and macrocytosis are conditions in which the red cells are respectively diminished and increased in size. They may occur in any form of severe anemia, but they are especially marked in pernicious anemia.

POIKILOCYTOSIS

Poikilocytosis, a condition in which the red cells are irregular in shape, is common in grave anemias, especially pernicious anemia.

NUCLEATED RED CELLS

Nucleated red cells (erythroblasts) are divided into three forms—normoblasts, macroblasts, and microblasts. The first resemble in size and color a normal red cell, the second are larger, and the third smaller. Nucleated red cells are not found normally in the circulating blood; they are present, however, in grave forms of anemia. A predominance of megaloblasts is very suggestive of pernicious anemia.

LEUKOCYTOSIS

Leukocytosis, or hyperleukocytosis, is an increase in the number of white cells, especially of the polymorphonuclear forms, in the peripheral blood. It occurs *physiologically* in the new-born, during digestion, in pregnancy and parturition, and after heavy exertion, cold bathing, and massage.

Pathologic leukocytosis is observed in the following conditions: (1) Inflammation. There is an absolute increase in the polymorphonuclear neutrophiles. (2) Infectious diseases. Most infections excite leukocytosis, but the condition is usually wanting in typhoid fever, malaria, measles, influenza, and miliary tuberculosis. In any infection in which the toxemia is intense or the resistance of the individual is slight, leukocytosis may also be wanting. (3) Malignant disease, if sufficiently extensive. (4) After hemorrhage. (5) After the administration of certain drugs, such as pilocarpin, antipyrin, salicylates, ergotin, and tuberculin. (6) In certain autointoxications, such as gout and uremia.

EOSINOPHILIA

An increase in the number of eosinophiles in the blood occurs in (1) helminthiasis (trichiniasis, uncinariasis, hydatid disease, etc.); (2) bronchial asthma; (3) certain skin diseases, notably eczema, pemphigus, psoriasis, and urticaria; (4) splenomedullary leukemia; (5) many postfebrile conditions.

LEUKOPENIA, OR HYPOLEUKOCYTOSIS

Leukopenia, or hypoleukocytosis, is the name applied to a deficiency in the number of leukocytes. It occurs in certain infections, particularly in those that do not produce leukocytosis, such as typhoid fever, malaria, and miliary tuberculosis; also in pernicious anemia and inanition.

LIPEMIA

Lipemia, the presence in the blood of minute fat-globules, may be noted in health. Abnormal quantities of fat may be found in the blood in diabetes, chronic nephritis, alcoholism, and pulmonary tuberculosis.

PARASITES IN THE BLOOD

The following parasites have been detected in the blood: Filaria sanguinis hominis, hematozoan of malaria, spirillum of relapsing fever, pneumococcus, bacillus of anthrax, typhoid fever, tetanus, tuberculosis, influenza, leprosy, glanders, bubonic plague, malignant edema, and diphtheria; the streptococcus, staphylococcus, meningococcus, gonococcus, trypanosoma, and colon bacillus.

OLIGOCHROMEMIA

Oligochromemia, or deficiency of hemoglobin, is usually proportionate to the reduction in the number of red cells; but there are two exceptions, namely, in chlorosis, in which disease the red cells may be reduced only 20 or 30 per cent., while the hemoglobin may be reduced 50 or 60 per cent., and in pernicious anemia, in which disease the blood-count is very low, while the corpuscles are relatively rich in hemoglobin.

The *color-index* represents the relation between the number of cells and the quantity of hemoglobin. In a patient having 2,500,000 red cells per cubic millimeter (50 per cent.) and 40 per cent. of hemoglobin, the color-index would be $^{40}/_{50} = 0.8$.

OLIGOCYTHEMIA

Oligocythemia, a diminution in the number of red cells, occurs in all forms of anemia, but it is especially intense after profuse or persistent hemorrhage, in pernicious anemia, in cancer, especially of the stomach, in some cases of advanced syphilis, and in certain forms of intestinal helminthiasis (hookworm and fish tapeworm).

ANEMIA

Definition.—A deficiency in corpuscular substance, *i.e.*, a deficiency in red corpuscles, in hemoglobin, or in both, with or without changes in the total volume of the blood (Cabot).

Varieties.—(1) Secondary anemia; (2) primary anemia.

Symptoms.—Any form of anemia may present the following symptoms: Pallor of the skin and mucous membranes, loss of strength, and, in severe cases, fibrile paroxysms.

Circulation.—A full, rapid pulse, unnatural pulsation of the cervical vessels, palpitation of the heart, a hemic murmur, a hum over the jugular vein, and slight dropsy, beginning in the feet. In severe forms there may be ecchymoses and bleeding from mucous membranes.

Respiration.—Hurried breathing.

Digestion.—Dyspepsia.

Nervous System.—Headache, vertigo, disturbed sleep, neuralgic pains, and tendency to syncope.

SECONDARY ANEMIA

Definition.—A secondary anemia is one that is symptomatic of some conspicuous underlying condition.

Etiology.—Secondary anemia usually results from one of three causes: (1) Insufficient nutriment entering the circulation (inadequate food, chronic gastritis, cancer of the pylorus, etc.) (2) Excessive demands upon the blood-making organs (overwork, hemorrhage, chronic diarrhea, etc.). (3) Action of parasites or toxic agents (malaria, lead, syphilis, uremia, etc.).

Symptoms.—In addition to the ordinary phenomena of anemia the blood-count reveals a decrease in the number of red cells and a proportionate deficiency in the percentage of hemoglobin. The number of polymorphonuclear leukocytes is often increased. In severe form, microcytes, macrocytes, and poikilocytes are present, and occasionally nucleated red cells.

Prognosis.—This depends on the cause.

Treatment.—This includes the removal of the cause, if possible; the adoption of hygienic measures; and the use of iron, arsenic, and general tonics.

PRIMARY ANEMIA

Definition.—A primary anemia is one that, in the present state of our knowledge, cannot be associated with any conspicuous underlying cause.

Varieties.—Pernicious anemia and chlorosis. Leukemia, Hodgkin's disease, and splenic anemia are also regarded as primary anemias by some authorities.

PERNICIOUS ANEMIA

(Progressive Pernicious Anemia)

Definition.—A grave form of anemia characterized by extreme oligocythemia, marked changes in the red blood-corpuscles, and decrease in the number of the polymorphonuclear neutrophiles.

Etiology.—In many cases no adequate cause is apparent. The disease usually appears about middle life, and is somewhat more frequent in males than in females. Forms of anemia closely resembling pernicious anemia may result from the action of intestinal parasites, especially the fish tapeworm (*Dibothriocephalus latus*) and the hookworm. The most plausible theory is that the disease is due to the hemolytic action of some poison produced within the body or the result of infection. According to W. Hunter, oral or gastric sepsis is the cause of the toxemia.

Pathology.—The skin has a lemon-yellow hue, the subcutaneous fat is often well preserved, and the muscles are unusually red. The organs are pigmented and fatty. Iron pigment is especially abundant in the outer zones of the hepatic lobules. Marked atrophy of the gastric mucosa is sometimes observed. The bone-marrow, as a rule, is dark red, soft, and contains large numbers of nucleated red cells, especially macroblasts (probably as the result of an attempt to compensate for the excessive hemolysis). The hemolymph glands are frequently enlarged, congested, and pigmented. The spleen is sometimes enlarged. In the majority of cases there is found degeneration of the spinal cord, especially in the posterior columns.

Symptoms.—The general symptoms are intense anemia, with its usual manifestations; a lemon-yellow tint to the skin; progressive weakness, without marked emaciation; attacks of moderate fever lasting a week or two; various gastro-intestinal disturbances, with paroxysms of epigastric pain; numbness, tingling, or other paresthesias in the extremities; and not rarely hemorrhages, especially into the skin or retina; slight or moderate enlargement of the liver and spleen, and symptoms referable to the spinal cord, most often those of tabes dorsalis.

The Blood.—The drop is pale and watery. There is a great reduction in the number of red cells, often to 1,000,000 or less; the hemoglobin is also reduced, but not proportionately (high color-index). The red cells usually show decided changes both in size (anisocytosis) and in shape (poikilocytosis). Nucleated red cells are more or less abundant. As a rule, the large forms (megaloblasts) predominate. The leukocytes are usually decreased, though the lymphocytes are relatively increased.

Diagnosis.—The *parasitic form* may be recognized by the occurrence of eosinophilia and the discovery of the parasites or their ova in the stools.

Cancer rarely produces such extreme oligocythemia, the color-index is not high, macroblasts are rarely present, and there is often leukocytosis.

Aplastic Anemia.—This rapidly fatal form of pernicious anemia especially affects young women. In contrast with the ordinary form of pernicious anemia the course of the disease is acute, there is much greater tendency to hemorrhages, the color index is low, nucleated red cells are usually absent, and the percentage of lymphocytes is relatively very high (75 to 90 per cent.). Anatomically, the bone-marrow is yellow instead of red, the erythroblastic tissue being replaced by fat.

Prognosis.—Pernicious anemia usually ends fatally in from one to three years. It is doubtful whether recovery ever occurs except in the parasitic forms. Remissions lasting from a few weeks to several months, or exceptionally two years or more, occur in the majority of cases.

Treatment.—Fresh air, rest, and a diet as liberal as the digestive power of the patient will permit are requisite. Warm salt baths and massage are valuable adjuvants to internal treatment. The teeth should receive careful attention. If there be gingivitis or pyorrhea alveolaris, antiseptic mouthwashes should be used at frequent intervals.

Arsenic is the most valuable drug. It may be given in the form of Fowler's solution, the dose being gradually increased from 2 or 3 to 15 or 20 minims three times a day. Iron is rarely of service. Bone-marrow is sometimes efficacious. Inhalations of oxygen have been recommended (Shattuck). Appropriate anthelmintic remedies should be given, of course, in the cases in which intestinal parasites are present. Digestive disturbances are often benefited by the administration of diluted hydrochloric acid and a bitter.

CHLOROSIS

(Green Sickness; Primary Anemia)

Definition.—A form of anemia occurring exclusively in young women and characterized by marked oligochromemia.

Etiology.—The disease usually occurs between the fifteenth and twenty-fifth years. Heredity, bad hygienic surroundings, and overwork are predisposing factors. The real cause of the disease has not been determined.

Pathology.—In some fatal cases imperfect development of the vascular and generative systems has been observed.

Symptoms.—In addition to the general symptoms of anemia the conspicuous features are a greenish hue of the skin; pallor and weakness without marked loss of flesh; dyspepsia with perversion of appetite; menstrual disorders, especially amenorrhea; and a tendency to hysteric outbreaks. The *blood changes* are characteristic. The number of red cells is moderately reduced (not often below 3,500,000); the hemoglobin, on the other hand, is greatly reduced—usually to below 50 per cent. There is no leukocytosis.

Complications.—Complications are uncommon, but peptic ulcer, hyperthyroidism, tuberculosis, or thrombosis of the cerebral sinuses or veins of the extremities may occur.

Prognosis.—The prognosis is good, but relapses are common.

Treatment.—Fresh air, sunlight, open-air exercise, and nourishing food are valuable aids in treatment. Very severe cases require complete rest in bed. If there be a good reaction, warm baths, followed by short cold douches, are efficacious. Iron is almost a specific. It is most frequently prescribed in the form of Blaud's pills, of which the dose is three pills, gradually increased to nine, a day.

Laxatives, preferably mild salines, rank next in importance to iron. Arsenic is distinctly less valuable than iron. Superacidity of the gastric juice is best treated by alkalis.

LEUKEMIA

(Leukocythemia)

Definition.—A disease characterized by an overgrowth of the leukoblastic tissues and an enormous increase in the number of circulating leukocytes.

Etiology.—The causes are obscure. More males are affected than females. The disease occurs most frequently in middle life. Heredity, malaria, syphilis, pregnancy, and traumatism are given as predisposing factors. An infectious origin has been suggested.

Varieties.—The bone-marrow, spleen, and lymph glands are all more or less involved in the hyperplasia, but according as the latter is of the myelocytic or of the lymphocytic type two forms of the disease are recognized: (1) myeloid (splenomedullary) leukemia; (2) lymphoid leukemia. The latter may be acute or chronic.

Pathology.—In the *myeloid form* the fat of the marrow, especially of the long bones, is largely replaced by a grayish-white or grayish-red tissue, which microscopically exhibits numerous myelocytes and closely related cells. The spleen and liver are much enlarged, the former from myeloid transformation (metaplasia), the latter from packing of the capillaries with leukocytes similar to those in the marrow and spleen.

Some of the lymph glands also show evidences of myeloid transformation.

In the *lymphoid form*, although enlargement of the lymph gland is usually the most striking feature, the changes are not limited to these parts of the hemopoietic system. The marrow is always affected and not rarely the spleen also shares in the process. The cellular transformation and proliferation, however, instead of being myelocytic, are lymphocytic, large or small lymphocytes preponderating according as the disease is acute or chronic.

Symptoms.—*Myeloid Leukemia.*—Progressive loss of flesh and strength and an increase in the size of the abdomen due to the enormous enlargement of the spleen are early manifestations. Pain in the splenic region is often experienced. The liver is moderately enlarged. Hemorrhages from the mucous membranes and into the skin, retina, or brain are common. Febrile attacks, usually lasting a week or two, occur in almost every case. The urine contains an excess of uric acid. Anemia is not an early symptom, but it always supervenes as the disease advances, and in the later stages pallor, dyspnea, digestive disturbances, edema, etc., may be conspicuous features. Hemorrhage or leukemic infiltration may cause dimness of vision if it occurs in the retina or optic nerve; deafness if it occurs in the labyrinth; or exophthalmos if it occurs in the orbit. Priapism is an occasional symptom.

The blood changes are characteristic. The number of leukocytes is enormously increased, the count usually ranging between 100,000 and 500,000. The increase is made up of all the normal cells and a large number, usually between 30 and 60 per cent., of myelocytes. The eosinophiles and mast cells are both relatively and absolutely increased. In advanced cases the number of red cells and the percentage of hemoglobin are considerably decreased.

Lymphatic leukemia is much less common. The general symptoms are much the same as in myeloid leukemia. The most conspicuous feature, however, is enlargement of the lymph glands—cervical, axillary, inguinal, etc. The spleen is more or less enlarged in almost every case. Enlargement of the tonsils is sometimes an early symptom suggesting tonsillitis. The striking change in the blood is the marked increase in the lymphocytes. The total leukocyte count usually ranges between 50,000 and 200,000, and the increase consists almost exclusively of lymphocytes. As a rule, small lymphocytes predominate in the chronic cases and large lymphocytes in the acute cases.

Atypical Leukemia.—Cases of lymphatic leukemia rarely occur in which there is no enlargement of the lymph glands, and, on the other hand, cases are occasionally observed in which the leukocyte count is below 30,000. Cases intermediate between myeloid leukemia and lymphoid leukemia have been reported. Chloroma is an atypical form of acute lymphatic leukemia in which green-tinted tumor-like masses (lymphoid infiltrations) penetrate the bones, especially those of the skull, and invade the surrounding tissues.

Diagnosis.—The ordinary chronic cases present no diagnostic difficulties, but atypical forms may go unrecognized and acute cases may readily be mistaken for certain infectious diseases, especially typhoid fever and septicemia, unless the blood be carefully studied. It must not be forgotten that under the influence of infections the leukocytes may become temporarily almost normal.

Prognosis.—The disease is always fatal. The average duration of the chronic forms is from two to three years. Remissions are not uncommon. Acute cases last from a few weeks to several months. Death may result from exhaustion or an intercurrent infection, such as tuberculosis, pneumonia, or septicopyemia.

Treatment.—An effort should be made to maintain the general nutrition by regulating the diet and attending to hygienic measures. Rest is often advisable. Among drugs, benzol appears to possess considerable potency, although it is not free from dangerous properties. From 10 to 20 drops, with equal amounts of olive oil, may be given in an emulsion three or four times a day. Blood-counts should be made at frequent intervals and the treatment should be suspended when the number of leukocytes has fallen to 30,000. Arsenic is also serviceable. The use of the *x*-rays is often followed by marked, though temporary, improvement. Operative treatment is of no avail.

HODGKIN'S DISEASE

(Pseudoleukemia)

Definition.—A comparatively rare disease characterized by enlargement of one or more groups of lymph-glands and progressive anemia.

Etiology.—The causes are unknown. It is most commonly seen in young adults of the male sex. Tuberculosis is sometimes associated with it, but the view that the disease is a form of tuberculosis has been abandoned. The reported finding of spirochetes has not been confirmed.

Pathology.—There is marked enlargement of the lymph-glands—cervical, axillary, inguinal, thoracic, retroperitoneal, etc. Microscopically, the changes consist of hyperplasia of the lymphoid cells with proliferation of the endothelial and reticular cells, formation of giant cells, and progressive fibrosis. It is doubtful whether a sharp line of demarcation can always be drawn between the lymphomas of Hodgkin's disease and malignant lymphomas (lymphosarcomas) or even some leukemic lymphomas. Nodules of lymphoid tissue are often found in the spleen, liver, bone-marrow, etc.

Symptoms.—The symptoms resemble those of lymphatic leukemia, but the blood changes are not characteristic. There is pronounced secondary anemia, with little if any increase in the number of leukocytes. The swollen lymph nodes are smooth, of varying consistency, painless, and almost never confluent. Symptoms, the result of local pressure—dyspnea, cough, cyanosis, edema, neuralgic pains, etc.—are often present. The spleen and liver are frequently enlarged. Periods of fever occur in almost every case. The disease is invariably fatal and the duration varies from a few months to several years.

Diagnosis.—*Tuberculous adenitis* may usually be distinguished by the tendency of the enlarged glands to fuse, to become adherent to the skin, and to suppurate and discharge. In doubtful cases the diagnosis must rest upon the microscopic examination of one of the enlarged glands excised for the purpose.

In *sarcoma* the growths develop, as a rule, more rapidly, are often painful, and show a marked tendency to fuse, to involve their capsules, and to invade adjacent structures. In some instances, however, the differential diagnosis may not be possible without a microscopic examination of an excised gland.

Treatment.—The treatment is that of leukemia.

HEMOPHILIA

Definition.—An hereditary constitutional defect characterized by immoderate and persistent hemorrhages after the most trivial injuries.

Etiology and Pathogenesis.—Nothing is known of the causes of the disease beyond the facts that it is strongly hereditary and familial, that it occurs chiefly in males, and that it is transmitted almost exclusively by unaffected females. The blood of hemophilic patients has a prolonged coagulation time; according to Sahli, this is due to a deficiency of thrombokinase produced in the walls of the blood-vessels and other tissues, but according to Howell it depends upon a decrease of prothrombin derived from the blood platelets.

Symptoms.—The chief symptom is the occurrence of profuse, at times uncontrollable, hemorrhages, usually following slight trauma, but not rarely developing spontaneously. Bleedings from the mucous membranes and into the subcutaneous tissues are very common, and hemarthrosis, sometimes resulting in inflammatory changes in the joints and suggesting rheumatic or tuberculous arthritis, is also frequent.

Prognosis.—The outlook is unfavorable in most cases. About 60 per cent. of all "bleeders" die before the eighth year, and only about 11 per cent. reach maturity. In some instances the tendency is outgrown.

Treatment.—This is chiefly protective and palliative. The most serviceable remedy is normal blood-serum (rabbit, horse, or man), of which from 20 to 30 c.c. may be given subcutaneously once or twice a day. Antidiphtheritic serum may be used, although it is less efficient than fresh serum. Calcium lactate (20 to 30 grains thrice daily) also exerts a beneficial effect, but only for a short time. Thyroid extract has proved efficacious in some instances. Local hemorrhages should be treated by rest, compression, and the application of hemostatics (fresh or dried blood-serum, adrenalin, gelatin, etc.). Hemophilic joints should be afforded absolute rest and after a few days subjected to gentle massage.

PURPURA

Purpura, or the occurrence of spontaneous hemorrhages into the skin and mucous membranes, is always a *secondary* condition, that is, a symptom of some underlying morbid state. Since, however, the cause of several purpuras is not known, it is customary to speak of a *primary* or *essential* type.

SECONDARY PURPURA

Secondary purpura occurs in the course of the following conditions: (1) *Acute infectious diseases.* A purpuric eruption is usually present in typhus fever and may occur also in cerebrospinal fever, septicopyemia (ulcerative endocarditis), measles, smallpox, etc. (2) *Cachectic states*, such as develop in carcinoma, scurvy, pernicious anemia, leukemia, chronic nephritis, old age, etc. (3) *Certain intoxications*, notably poisoning by iodids, antipyrin, salicylic acid, quinin, and mercury. Apart from drug poisoning, snake bite and jaundice may also cause purpura. (4) *Venous stasis.* Purpura from this cause may occur about tight bandages or after epileptic seizures or the paroxysms of whooping-cough. (5) *Nervous disorders.* The purpuras occasionally observed in hysteria (stigmata) and rarely in association with neuralgia, sciatica, and the lightning pains of tabes dorsalis belong in this group.

PRIMARY PURPURA

Etiology and Pathogenesis.—The cause of primary purpura is unknown. The disease occurs most frequently in the second and third decades and is more common in males than in females. The view that it is caused by the action of some unknown toxin on the capillaries or on the blood itself has many advocates. The number of blood platelets is greatly diminished; the blood coagulates within the normal time, but the clot contracts slowly and imperfectly.

Symptoms.—Several varieties are recognized, but the barrier separating them is not very sharply defined.

Purpura Simplex.—In this form the chief feature is the appearance in successive crops of small hemorrhagic spots on the extremities, especially the legs. Pains in the joints are frequently noted, and when these are pronounced the condition is usually designated *purpura rheumatica* (Peliosis rheumatica of Schönlein). Urticaria is also common. The duration is from a few days to several months.

Henoch's Purpura.—This form is characterized by recurring outbreaks of purpura and attacks of abdominal pain, often accompanied by vomiting and diarrhea (exudative erythema of the bowel). The discharges are not rarely bloody. Arthritis is common. Erythema, urticaria, and localized edema are noted in many cases. Nephritis is a frequent complication. The condition occurs most often in early life and seems to be closely related to angioneurotic edema. The duration of the entire illness is from two or three weeks to as many months.

Purpura Hemorrhagica (Morbus Maculosus of Werlhof).—In this form there is bleeding from the mucous membran especially the nose, mouth, and genito-urinary tract, as well as into the skin. Fever is present in more than half of the cases. Arthritis and nephritis are not infrequent. Anemia soon develops in severe cases. The average duration is from four to six weeks. Relapses are common. In rare instances the disease assumes a malignant character (*purpura fulminans*) and ends fatally within two or three days; on the other hand, it occasionally becomes chronic and lasts for many years.

Associations.—Urticaria, erythema multiforme, erythema nodosum, localized edema (angioneurotic edema), and nephritis are of frequent occurrence in all forms of primary purpura. Nephritis is especially common in Henoch's purpura and may prove fatal. Endocarditis, pericarditis and cerebral hemorrhage are rare complications.

Diagnosis.—*Scurvy* may be distinguished by a history of dietetic errors, by the spongy state of the gums, and by the brawny induration of the muscles. In *hemophilia* the hemorrhagic tendency is hereditary and congenital. *Secondary purpura* may usually be differentiated by the history and the other symptoms of the primary condition. Care must be taken not to mistake the abdominal crises of Henoch's purpura for intussusception or appendicitis.

Prognosis.—Recovery is the rule, except in the severe forms. In fatal cases death may be caused by cerebral hemorrhage, nephritis, or exhaustion.

Treatment.—The patient should be put to bed and given light nourishing food. The special remedies most worthy of trial are calcium lactate (15 grains thrice daily), arsenic in increasing doses, oil of turpentine, and gelatin. Iron is useful during convalescence.

ERYTHREMIA

(Vaquez' Disease)

Erythremia is a rare affection characterized by persistent redness or cyanosis of the skin, polycythemia (7 to 13 millions per c.mm.), and marked enlargement of the spleen. Less common symptoms are high blood-pressure, vertigo, headache, albuminuria and ascites. Hemorrhage is not infrequent, especially cerebral hemorrhage. As a rule, the disease lasts many years, a fatal event ultimately occurring from heart failure or hemorrhage. Hyperactivity of the erythroblastic tissues of the bone-marrow is believed to be the basis of the disorder. In making the diagnosis one must exclude other causes of persistent cyanosis, such as chronic cardiac and pulmonary disease, drug poisoning (acetanilid, antipyrin, and other coal-tar products), the rare forms of chronic enteritis in which cyanosis occurs probably from the absorption of nitrites or hydrogen sulphide from the intestines (enterogenous cyanosis), and tuberculosis of the spleen. Benefit has resulted from venesection, *x*-rays, and saline purges in some cases.

DISEASES OF THE DUCTLESS GLANDS

ADDISON'S DISEASE

Definition.—A rare disease due to inadequacy of the adrenal glands or other parts of the chromaffin system and characterized by extreme muscular and cardiovascular weakness, pigmentation of the skin and mucous membranes, and gastro-intestinal irritability.

Etiology.—The disease occurs most frequently between the ages of twenty and fifty years, and is more common in males than in females. The causes of tuberculosis in general favor its development.

Pathology.—The usual lesion is tuberculosis (fibrocaseous) of the adrenals. Occasionally there is simple atrophy, fibrosis, or tumor. In some instances degenerative changes are found in the adjacent sympathetic trunks or ganglia either in connection with, or in the absence of, changes in the adrenals.

Symptoms.—Profound muscular weakness, developing without obvious cause and unaccompanied by any corresponding emaciation, is an early symptom. The arterial pressure is usually very low and in advanced cases attacks of syncope are of frequent occurrence. Brownish pigmentation or bronzing of the skin, almost universal, but especially marked on the exposed parts and genitalia, around the nipples and navel, and wherever the skin has been irritated or compressed, appears sooner or later in nearly every case. In many cases the mucous membranes are also affected. Gastro-intestinal irritability, manifested by anorexia, epigastric discomfort or pain, and attacks of vomiting or diarrhea, is also common. The blood shows the usual changes of secondary anemia.

Diagnosis.—This is difficult until the symptoms are well developed. Other forms of pigmentation, such as that occurring in chronic jaundice, malarial melanemia, melanotic sarcoma, hemochromatosis (bronzed diabetes), pregnancy, malignant disease within the abdomen, pellagra, argyria, chronic arsenic poisoning and vagabond's disease (discoloration resulting from uncleanly habits and exposure), etc., must be excluded.

Prognosis.—The disease is probably always fatal, and usually lasts from a few months to several years. The course is not uniform but marked by remissions and exacerbations. Death is usually due to asthenia, but it may occur from syncope or a toxic condition (convulsions, delirium, coma).

Treatment.—The general treatment includes rest, a nutritious but easily digestible diet, and the administration of tonics. In some cases temporary good effects have followed the use of the extract of the suprarenal gland in doses of from 15 to 120 grains thrice daily.

EXOPHTHALMIC GOITER

(Hyperthyroidism; Graves' Disease; Parry's Disease; Basedow's Disease)

Definition.—A disease characterized by enlargement of the thyroid gland, marked prominence of the eyeballs, tachycardia, and muscular tremors.

Etiology.—The disease most frequently develops in the third or fourth decade, and affects women much more often than men. In many instances it is consequent upon acute disease, pregnancy, the menopause, or profound mental or emotional strain. In some cases it supervenes on ordinary goiter.

Pathology.—The pathogenesis of Graves' disease is still undetermined. Excessive functional activity of the thyroid gland (hyperthyroidism) is undoubtedly the basis of the disorder, but the initiating cause is unknown. The essential changes in the gland are proliferation of the epithelial cells and fibrous tissue with a diminution of the colloidal material.

Symptoms.—*Cardiac Phenomena.*—Acceleration of the pulse (100 to 150) and palpitation are constant features. Both are intensified by excitement. Hypertrophy of the heart may ultimately ensue from overaction. A soft systolic murmur is frequently heard at the apex.

Ocular Phenomena.—Exophthalmos, or protrusion of the eyeballs, usually bilateral, is an obtrusive symptom. Accompanying it there are often other ocular changes, such as lagging of the upper eyelids in downward movement of the eyeballs (von Graefe's sign); a peculiar staring look, due to widening of the palpebral fissure (Dalrymple's sign); infrequent and incomplete reflex winking (Stellwag's sign); and insufficient power of convergence for near objects (Möbius' sign). As a rule, vision is not disturbed.

Thyroid Phenomena.—Enlargement of the thyroid may be the last symptom to appear. One or both lobes of the gland may be affected. Palpation often detects pulsation and a purring thrill, and auscultation, a soft systolic bruit.

Nervous Phenomena.—A fine muscular tremor, most pronounced in the extremities, is an early symptom. Abnormal irritability and extreme restlessness are characteristic of the disease. Vasomotor disturbances, such as excessive flushing and sweating, urticaria, and local edema are frequently observed. Mania or melancholia occasionally supervenes.

General Symptoms.—As the disease progresses, weakness, emaciation, and anemia usually become pronounced. Attacks of vomiting and of serous diarrhea are common. Moderate fever is an occasional symptom. There may be glycosuria and albuminuria.

Diagnosis.—Only the incomplete forms (*formes frustes*) are likely to escape recognition. Tachycardia is never absent, but neither goiter nor exophthalmos is an essential symptom.

Prognosis.—The disease usually runs a protracted course, extending over many years. Some patients (probably 30 per cent.) completely recover, many improve, and a few die within a few weeks or a few months (acute form). Relapses are common. Occasionally myxedema supervenes. Death may result from heart failure (sudden or gradual), intercurrent disease, or exhaustion.

Treatment.—The general nutrition should be improved by rest, freedom from excitement, a generous, readily digestible diet, and hydrotherapy. In severe cases absolute rest in bed is an essential point in the treatment. Applications of cold, by means of Leiter's tubes or ice-bags, to the precordium lessen the palpitation.

Belladonna is undoubtedly of value in many cases. It should be given in ascending doses until some dryness of the throat is produced. If the circulation is feeble, digitalis may be found of service; on the other hand, if the heart is strong, better results may be obtained with aconite or veratrum viride. If anemia exists, iron and arsenic are useful. Bromids are sometimes of service in controlling nervous symptoms. Starr has observed marked improvement from the use of sodium glycerophosphate in doses of 20 grains three or four times a day. The consensus of opinion is adverse to the use of thyroid extract. Fresh milk, serum, and dried blood of thyroidectomized animals, and "antitoxic" serum have been used with some success. Electricity has occasionally proved efficacious. Surgical treatment (partial resection of the gland or ligation of two or three of the thyroid arteries) is comparatively safe in expert hands, and offers good hope of cure or great amelioration.

MYXEDEMA; HYPOTHYROIDISM

Definition.—A chronic disease due to a lack of thyroid secretion and characterized by brawny thickening of the subcutaneous tissues, loss of energy and mental deterioration.

Etiology.—The disease is much more frequent in women than in men. It is occasionally hereditary or familial. It usually develops between the ages of twenty and fifty years. The basic condition is atrophy of the thyroid gland, but the cause of this morbid change is unknown.

A congenital form of myxedema is observed in *cretinism*, and an analogous condition (*operative myxedema* or *cachexia strumipriva*) frequently follows total extirpation of the thyroid gland.

Symptoms.—It is manifested by a gradual swelling of the subcutaneous tissues, particularly of the face, supraclavicular regions, and hands. Unlike edema, the parts do not pit on pressure, but are firm and elastic. The skin is dry and harsh. The hair becomes brittle and falls out. The thyroid gland is atrophied. A peculiar slowness in thought, speech, and movements is a characteristic symptom. Eventually dementia may supervene. The temperature of the body is subnormal and the pulse is infrequent. There is impairment of the special senses. Sensory phenomena, such as undue sensitiveness to cold, neuralgic pains, and formication are common. Albuminuria and glycosuria sometimes occur. The sexual functions are depressed.

In *cretinism*, which may be endemic or sporadic, there is arrested development, physical and mental, with changes in the skin like those in myxedema and a characteristic deformity of the bones and soft parts. The head is large, the features are coarse and bloated, the expression is stolid or idiotic, the trunk and limbs are short and thick, the abdomen is protuberant, the sexual organs are infantile, and the skin is rough and dry.

The symptoms of *cachexia strumipriva* are much the same as those of spontaneous myxedema in the adult.

Diagnosis.—Mild cases are likely to be overlooked unless examined very carefully. *Chronic nephritis* may give rise to confusion, but in this disease the swelling pits on pressure, the skin is not thickened and indurated, the hair does not fall, and there is no peculiar slowing of the mental processes.

In *acromegaly* the face is elongated, the chin is prominent, kyphosis is common, the hair grows well, and the hypertrophy affects the bones as well as the soft parts.

Cretinism is sometimes confused with mongolian idiocy and with achondroplasia. In the *mongolian idiot*, however, the eyes are obliquely set, the subcutaneous tissues are not thickened, and there is restlessness and vivacity rather than inertness and stolidity. In *achondroplasia* the limbs are very short, with the hands and feet attached almost directly to the trunk, the intellect is good, and the skin is natural.

Prognosis.—Marked improvement or even symptomatic cure is possible provided appropriate treatment is begun early and continued throughout life. In the absence of specific therapy the tendency of the disease is to become progressively worse.

Treatment.—As patients with myxedema are extremely susceptible to low temperatures, they should be warmly clad and protected from exposure to cold. Residence during the winter in a warm, sunny climate is desirable. Warm baths are often beneficial. Modern treatment consists in the administration of extract of sheep's thyroid (from 1 to 5 grains thrice daily, the final dose depending upon individual need or tolerance).

ACROMEGALY

Definition.—A chronic disease depending upon changes in the pituitary body and characterized by abnormal growth, especially of the hands, feet, and face.

Etiology.—The cause is unknown. The disease is more common in males than in females, and usually begins between the twentieth and fortieth years.

Pathology.—In the vast majority of cases the pituitary body is the seat of hyperplasia or tumor formation (benign or malignant). Other ductless glands, especially the thyroid and the thymus, may also be enlarged. Hypertrophic changes are observed in the bones and subcutaneous tissues and in some cases also in the heart, liver, and spleen. The symptoms are probably due to hyperactivity of the pituitary body, especially of the anterior lobe (hyperpituitarism).

Symptoms.—Gradual enlargement of the hands, feet, and face is the most conspicuous symptom. The face is elongated, the nose large and bulbous, the lower jaw massive and prominent (prognathism), and the lips and tongue are thick. The hands are large and spade-like, and the fingers sausage-shaped. The feet are affected in a similar way. Kyphosis is almost invariably present. Impotence in the male and amenorrhea in the female are early manifestations. Symptoms of gross intracerebral disease, especially headache and defect in sight (hemianopsia), often appear. Muscular weakness, mental hebetude, excessive thirst, and glycosuria are frequently noted in advanced cases. Cardiovascular disease, diabetes, myxedema, and exophthalmic goiter are not uncommon associations. The disease is progressive but usually lasts many years. If the lesion of the hypophysis is malignant, however, the duration may be only two or three years.

Diagnosis.—This is rarely difficult except in the earliest stages. In *Paget's osteitis deformans* the long bones are especially involved and are not only enlarged but are considerably deformed, and the face has a peculiar triangular shape. The distinctive features of *myxedema* have already been stated (see p. 183).

Treatment.—Medical treatment is unsatisfactory. Thyroid extract and thymus extract have been used with apparent benefit in a few cases. Removal of the pituitary tumor has been successfully accomplished by Cushing and others in a number of instances.

HYPOPITUITARISM

The condition first described by Frölich under the name of **dystrophia adiposo-genitalis** and characterized by general obesity, hypoplasia or atrophy of the sexual organs, a lack of pubic hair, somnolence, and a high tolerance to carbohydrates is apparently due to insufficiency of the hypophysis, although it is not improbable that other glands of internal secretion may also be concerned in the process. Closely related conditions are:

(1) **Adiposis Dolorosa (Dercum's Disease).**—In this disorder there are more or less symmetrical deposits of fatty masses in various parts of the body, attended or preceded by pain, and frequently associated with asthenia and psychic disturbances.

(2) **Ateliosis.**—The chief features of this condition, which is nearly the antithesis of acromegaly, are a childish facial appearance, diminutive stature, with short slender limbs, ill-developed muscular prominences, small jaw bones, a thin piping voice, low arterial tension, and scanty urination.

(3) **Idiopathic Infantilism (Lorain Type).**—In this disorder the stature is diminutive and there is imperfect development of the sexual organs and the secondary sexual characteristics, but the outlines of the body are those of a well-formed adult.

(4) **Progeria.**—This is a form of infantilism associated with premature senility and sclerotic changes in the cardiovascular system.

TETANY

Definition.—A rare disease characterized by bilateral tonic, intermittent, or continuous spasms, especially of the extremities, and increased excitability of the nerves and muscles to mechanical and electrical stimulation.

Etiology.—Postoperative and non-operative cases are observed. Postoperative tetany sometimes follows operations upon the thyroid (removal or destruction of the parathyroids). Non-operative tetany in adults is most commonly seen as a complication of gastrectasis, but it may occur in other severe gastro-intestinal affections, in pregnancy or with lactation, in acute infectious diseases, or in poisoning from certain substances, such as chloroform, ergot, etc. In Austria it is sometimes endemic, chiefly among handworkers—shoemakers, tailors, etc. In young children it is usually associated with rickets. The researches of MacCallum and Voegtlin indicate that tetany is due to a lack of calcium in the blood, the result of parathyroid insufficiency.

Symptoms.—The patient is seized with bilateral tonic spasms, beginning in the hands and feet and spreading upward. In many instances only the hands are affected. The muscles of the trunk and face are seldom involved. The spasms usually occur in paroxysms lasting from a few minutes to an hour or two, but in very severe cases they may be more or less continuous. They are sometimes attended by pain. Laryngismus stridulus is not uncommon in children. The mind is usually unaffected. Occasionally the cramps are accompanied by slight fever and edema. Even when the contractions have ceased they may be reproduced by the application of pressure over the nerve trunks or vessels of the affected member (*Trousseau's sign*). Sometimes a mere tap over the nerve is sufficient to excite spasm (*Chvostek's sign*). The electrical excitability of the motor nerves is also increased (*Erb's sign*). Percussion or electrical stimulation of sensory nerves not infrequently calls forth abnormally intense sensations (*Hoffmann's sign*). Very mild attacks (*latent tetany*) may consist of paresthesia, muscular irritability, and slight stiffness without spasm.

Diagnosis.—In *tetanus* a source of infection is usually found; trismus or lockjaw appears early, and the muscles of the back are more contracted than those of the limbs.

Hysteria may be distinguished from tetany by the history, the emotional disturbances, the unilateral character of the spasms, and the absence of Trousseau's sign.

Prognosis.—The attacks may occur at intervals of hours or days, and the entire disease may last from a few days to several months. Relapses are common. The outlook, generally speaking, is good, although the mortality is high in tetany arising in gastrectasis.

Treatment.—The cause should be sought for and removed if possible. Calcium salts (15 grains of the lactate four times a day) and parathyroid preparations are the most serviceable remedies. In cases of emergency calcium lactate (500 c.c. of a 5 per cent. solution in normal saline solution) should be given intravenously with subcutaneous injections of emulsion of freshly prepared parathyroid gland. Bromids, chloral, or morphin may be used as adjuvants. Warm baths are beneficial. Surgical intervention is indicated in gastrectasis.

PRIMITIVE SPLENOMEGALY WITH ANEMIA

(Banti's Disease; Splenic Anemia)

This is a chronic disease of unknown origin, characterized by progressive enlargement of the spleen, anemia of the secondary type, leukopenia, a marked tendency to hemorrhages, and, in certain cases after the lapse of years, cirrhosis of the liver, with ascites and slight jaundice (Banti's disease).

The splenic enlargement is very pronounced and for many years may be the only conspicuous feature. The blood-picture is, as a rule, that of moderate anemia (3,000,000 to 4,000,000 erythrocytes per c.mm.), with a low color-index and marked leukopenia. The hemorrhages are usually from the stomach or bowel and are often profuse. Cirrhosis of the liver, with enlargement or atrophy of the organ, ascites, jaundice, etc., is a late phenomenon, and the one to which Banti directed special attention. The pathologic changes in the spleen consist of an overgrowth of connective tissue, hyperplasia of the endothelial cells, and endophlebitis of the splenic vein. The portal vein may also be affected. The disease is remarkably chronic, often lasting ten years or longer. Done at the proper time, splenectomy frequently effects a complete cure. The mortality of the operation in 118 cases was 19 per cent.

OTHER CHRONIC ENLARGEMENTS OF THE SPLEEN

Chronic enlargement of the spleen other than that occurring in Banti's disease is observed in the following conditions:

(1) **Tumors and Cysts.**—Sarcoma, endothelioma, echinococcus cysts, etc. Many observers regard the Gaucher type of splenomegaly as a neoplasm. A characteristic feature of this disease is the presence of peculiar large cells (20–40 microns in diameter) in the spleen, lymph-glands, and bone-marrow. Clinically, it closely resembles Banti's dise but is distinguished by its family occurrence and predilectio childhood, by the early appearance of a brownish-yellow discol

(non-icteric) of the face and hands, and wedge-shaped thickening of the conjunctiva, and by the absence of ascites.

(2) **Diseases of the Blood-making Organs.**—Leukemia, pernicious anemia, erythremia, etc. The definite blood-changes in these conditions are characteristic.

(3) **Chronic Infections.**—Malaria, syphilis, tuberculosis, Kala-azar, etc. Syphilis is probably the commonest cause of infantile splenomegaly. Massive tuberculosis of the spleen may closely resemble Banti's disease.

(4) **Disorders of Nutrition.**—Rickets, amyloid disease, certain obscure gastro-intestinal intoxications, etc.

(5) **Cirrhosis of the Liver.**—In the later stages the clinical picture may be like that of Banti's disease. The early appearance and marked prominence of the hepatic symptoms and, in Hanot's cirrhosis, the occurrence of leukocytosis are important diagnostic features.

(6) **Passive congestion of the spleen,** the result of chronic heart disease, primary pylephlebitis, and other obstructive disorders of the portal circulation.

(7) **Chronic Family Acholuric Jaundice** (Splenomegalic Jaundice).—This condition is described on p. 95.

(8) **Anemia in Infancy and Early Childhood.**—This includes a large ill-defined group of cases, some of which are little understood. In many cases the anemia and splenic enlargement are clearly secondary to such diseases as syphilis, rickets, scurvy, etc., but in others the cause is not obvious. In the latter group are included examples of Banti's disease or Gaucher's disease, acholuric jaundice, aberrant forms of leukemia, and the anemia pseudoleukemia infantium of von Jaksch.

DISEASES OF THE CIRCULATORY SYSTEM

INSPECTION

INSPECTION serves to determine the position, force, and extent of the apex-beat and to detect any unnatural prominences in the region of the heart or aorta and any abnormal centers of pulsation.

The Apex-beat.—In healthy adults the apex-beat is in the fifth left intercostal space, about half an inch internal to the midclavicular line. In young children it is commonly in the fourth interspace, and in old persons it is often as low as the sixth interspace.

The Effect of Respiration and Position upon the Apex-beat.—The location and force of the apex-beat are modified by the posture of the patient and the stage of the respiratory act. In the recumbent position the apex-beat may be elevated half an inch or more, and when the body is inclined to the left, the heart being a more or less movable organ, the beat may be detected in the midclavicular line, or even some distance to its outer side.

During forced inspiration the beat may become imperceptible or, if such is not the case, it may be found some distance below its usual place, on account of the upward movement of the ribs in the inspiratory act. During forced expiration the beat becomes more forcible and its position elevated on account of the descent of the ribs which occurs in expiration.

In view of the influence exerted by respiration and position upon the apex-beat, the patient, as a rule, should be examined in the erect or sitting posture, while breathing quietly.

Displacement of the Apex-beat.—*Displacement to the left* may result from:

1. Hypertrophy or dilatation of the heart (down and to the left).

2. Chronic diseases of the left lung and pleura, associated with retraction—as fibroid phthisis and pleural adhesions.

3. Abdominal tumors and effusions (up and to the left).

4. The pressure of a pleural effusion on the right side (up and to the left).

Displacement to the right may be caused by:

1. Chronic disease of the right lung or pleura associated with retraction.

2. Pressure of a pleural effusion on the left side.

3. Transposition of the viscera.

Displacement downward may result from:

1. Hypertrophy or dilatation of the heart, chiefly of the left ventricle.

2. Pressure of solid growths in the upper mediastinum.

3. Aneurysm of the aortic arch.

Deformity of the chest from spinal curvature may also cause considerable displacement of the heart.

Changes in Force and Extent of the Apex-beat.—*The force and extent of the pulsation may be increased* by:

1. Hypertrophy of the heart.

2. Forcible action of the heart caused by emotional or physical excitement, reflex irritation, drugs, Graves' disease, etc.

3. Thinning of the chest-walls and shrinking of the lungs, as in pulmonary tuberculosis.

The strength of the apex-beat may be lessened by:

1. Degeneration or dilatation of the heart.

2. Pericardial effusion.

3. Emphysema.

4. Collapse or shock.

Abnormal Centers of Pulsation.—*Epigastric pulsation* may result from:

1. Excited action of the heart from any cause.

2. Enlargement of the right ventricle.
3. A pulsating aorta, noted in certain nervous and anemic patients.
4. Low position of the heart from arteriosclerosis.
5. Aneurysm of the abdominal aorta.
6. Tumors of the left lobe of the liver resting on the aorta.

Pulsation at the base of the heart may result from:

1. Aneurysm of the aortic arch.
2. Cardiac hypertrophy.
3. Shrinking of the lungs, as in tuberculosis.

Pulsation in the left axillary region may result from:

1. Large right-sided pleural effusions.
2. A tense, purulent effusion in the left pleural sac (pulsating empyema).
3. Aneurysm.

Unnatural pulsation in the carotids may result from:

1. Excitement of the heart from any cause.
2. Exophthalmic goiter.
3. Anemia.
4. Valvular disease, especially aortic regurgitation.
5. Aneurysm or dilatation of the vessels.

Jugular Pulsation.—The jugular vein often becomes distended in forced expiration and coughing. Inspiratory overfulness (Kussmaul's sign) of the jugular vein is sometimes noted in adherent pericardium.

A positive or systolic venous pulsation is observed in tricuspid regurgitation.

A pulsation may be transmitted to the jugular vein from the underlying carotid, but this false pulsation will still continue when light pressure is made on the vein at the root of the neck, while the true venous pulse will cease.

Precordial Prominence.—*Unnatural prominence of the precordium* may result from: (1) Enlargement of the heart; (2) pericardial effusion; (3) aortic aneurysm; (4) rachitic deformity.

PALPATION

This serves to determine the position, force, extent, and rhythm of the apex-beat, and also to detect the presence of thrills.

A *thrill* is a vibratory sensation likened to that received when the hand is placed on the back of a purring cat. Thrills at the base of the heart may result from aortic stenosis, atheroma of the aorta, aneurysm, and congenital heart disease (pulmonary stenosis and patency of the ductus arteriosus).

A systolic thrill may sometimes be felt at the apex in mitral regurgitation. A presystolic apical thrill is almost pathognomonic of mitral stenosis.

PERCUSSION

This serves to determine the shape and extent of the cardiac dulness.

The normal area of superficial or absolute percussion-dulness (the part uncovered by lung) is detected by light percussion, and extends from the fourth left costosternal junction to the apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum, and thence up the left border of the sternum.

The normal area of deep percussion-dulness (the heart projected on the chest-wall) is detected by firm percussion, and extends from the third left costosternal articulation to the apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum, and thence up the right border of the sternum to the third rib. The lower level of the cardiac dulness fuses with the liver dulness, and can rarely be determined by percussion.

The area of cardiac dulness is increased in: (1) Hypertrophy and dilatation of the heart. (2) Pericardial effusion. It is apparently increased in shrinking of the lungs, as in tuberculosis.

The area of cardiac dulness is diminished in: (1) Emphysema. (2) Pneumothorax. (3) Pneumopericardium (rare). (4) Gaseous distention of the stomach.

AUSCULTATION

This serves to determine the quality, intensity, and rhythm of the heart-sounds, and to detect the presence of any adventitious sounds, such as murmurs. The two sounds heard over the heart have been represented by the syllables, "lubb, tup." The first sound (*systolic*) results from the muscular contraction of the heart and the closure of the auriculoventricular valves, and is synchronous with the apex-beat and carotid pulse. This sound is prolonged and dull. After the first sound there is a short pause, and then follows the second sound (*diastolic*), which results from the closure of the aortic and pulmonary valves. This sound is short and high-pitched. After the second sound a longer pause follows before the first is again heard.

The Intensity of the Heart-sounds.—*Both sounds are accentuated* in: (1) Excitement of the heart from any cause. (2) Anemia. (3) Cardiac hypertrophy. (4) Subjects with thin chest-walls. (5) Consolidation of the lung, as in tuberculosis and pneumonia.

Accentuation of the aortic second sound results from: (1) Hypertrophy of the left ventricle. (2) High arterial tension, as in chronic interstitial nephritis with arteriosclerosis. (3) Aortic aneurysm.

Weakening of the aortic second sound indicates weakness of the left ventricle.

Accentuation of the pulmonary second sound results from: (1) Pulmonary obstruction, as in emphysema, pneumonia, and the congestion of the lungs following mitral disease. (2) Hypertrophy of the right ventricle.

Weakness of the pulmonary sound indicates failure of the right ventricle, and, occurring in diseases in which it should be accentuated, is of grave omen.

Weakness of the mitral sound is noted in: (1) General obesity. (2) General exhaustion. (3) Degeneration or dilatation of the heart. (4) Pericardial or pleural effusion. (5) Emphysema.

Alteration in the Rhythm of the Heart-sounds.—*Reduplication of the Diastolic Sounds.*—This is probably due to a lack of synchronism in the closure of the aortic and pulmonary valves. It is frequently noted in health at the end of a long inspiration. Pathologic reduplication may occur whenever the pressure in either the pulmonary circulation or the peripheral arteries is abnormally increased. It is a common sign in mitral stenosis, emphysema, arteriosclerosis, and pericarditis.

Embryocardia.—This term is used to indicate a rhythm that resembles that of the fetal heart. The pauses between the sounds are of equal length, and the two sounds are exactly alike. Embryocardia indicates great enfeeblement of the heart and may be observed in exhausting fevers and in cardiac failure from any cause.

Gallop or Cantering Rhythm.—This suggests the hoof-beats of a galloping horse. One of the cardiac sounds is doubled and the diastolic pause is shortened. It indicates extreme weakness of the heart.

Adventitious Sounds.—*Murmurs* are abnormal sounds produced in the heart or blood-vessels. They may result from: (1) Obstruction or regurgitation at the valves in consequence of valvular endocarditis. (2) Dilatation of the ventricle or relaxation of its walls, in consequence of which the auriculoventricular valves become relatively insufficient. (3) Roughening of the valves or of the intima of the great vessels. (4) Aneurysm (bruit). (5) Anemia (hemic murmur).

Exocardial murmurs are adventitious sounds of cardiac origin produced in the pericardium (pericardial friction-sound) or in the pleura or lung, adjacent to the heart (pleuropericardial friction-sound and cardiorespiratory murmur).

Pericardial Friction-sound.—This is an adventitious sound produced in pericarditis by roughening of the serous membrane. It is a harsh, grating, to-and-fro sound, quite superficial, often intensified by pressure with the stethoscope, and generally heard best in the fourth interspace near the sternum. It often varies in intensity from hour to hour, and is rarely transmitted beyond the precordial region.

Pleuropericardial Friction-sound.—This is a sound closely resembling the pericardial friction-sound, but produced by inflammation of that part of the pleura that overlaps the heart. It is intensified by deep inspiration, and often disappears when the breath is held during expiration.

Cardiorespiratory Murmur.—This is a rare adventitious sound, produced by the rhythmic expulsion of air from the lappet of lung covering the heart by the cardiac contractions. The exact condition under which it occurs is not known. It is usually heard best at the end of inspiration, and is nearly always systolic in time. It is greatly modified by position, deep breathing, coughing, and holding the breath.

Aneurysmal Murmur, or Bruit.—In a certain proportion of cases a murmur is heard in aneurysm. It is systolic in time, heard with greatest intensity over the sac of the aneurysm, and transmitted into the vessels of the neck. There is nothing in the character of the murmur to suggest its origin.

Hemic Murmurs.—Hemic murmurs have the following characteristics: They are soft and blowing in character; they are usually systolic in time; they are usually heard best over the pulmonary area; they are associated with evidences of anemia; they are not accompanied by signs of cardiac enlargement; they are often associated with a continuous hum in the veins of the neck; and they are more affected by deep breathing, position, and exercise than the murmurs of organic disease.

THE PULSE

The average frequency of the pulse in the healthy adult at rest is between 70 and 80. In new-born infants it is between 130 and 140, and in young children between 90 and 100.

Increased Frequency of the Pulse (Tachycardia).—Habitual frequency is sometimes noted in health. The frequency may be temporarily increased by erect posture, excitement, eating, and the use of stimulants.

Abnormal frequency may be due to—(1) Pyrexia. The pulse usually bears a definite relation to the temperature, but in certain diseases, as scarlet fever and septicemia, it may be disproportionately rapid, and in others, like meningitis and yellow fever, it may be disproportionately slow. (2) Exophthalmic goiter. (3) Organic heart disease. (4) Severe anemias. (5) Reflex irritation, as in ovarian or uterine disease. (6) Essential paroxysmal tachycardia, the result of extrasystoles caused by hyper-excitability of some focus in the auricle, auriculoventricular bundle, or ventricle. (7) Action of certain drugs—belladonna, nitrites, thyroid extract, etc. (8) Rheumatoid arthritis (Sansom).

Infrequency of the Pulse (Bradycardia).—*Physiologic infrequency* is noted after fasting, sometimes in the puerperium, and habitually in certain persons (50 to 60 a minute).

Pathologic infrequency is observed in many conditions, notably—(1) In certain forms of organic heart disease (especially marked in lesions of the auriculoventricular bundle causing full heart-block). (2) In many toxemias—jaundice, uremia, myxedema, plumbism, digitalis-poisoning. (3) From pressure at the base of the brain sufficient to irritate the vagus, as in beginning meningitis, tumor, etc. (4) At the close of febrile diseases, as typhoid fever, pneumonia, etc. (5) In certain painful affections of the digestive tract. (6) In cachectic states.

Arhythmia.—Alterations in the normal cardiac rhythm may depend upon disturbances of the nervous mechanism of the heart (*neurogenic arhythmia*) or upon disturbances arising within the heart muscle itself (*myogenic arhythmia*). If the irregular beats occur in a definite order the condition is termed *allorhythmia*. Extreme irregularity without any discernible order in the arrangement of the beats is frequently spoken of as *delirium cordis.*

Neurogenic Arhythmia may be caused by strong emotions, by various toxic agents (tobacco, coffee, alcohol, bacterial poisons), by meningitis or cerebral lesions causing increased intracranial pressure, and by various impressions emanating from the organs, especially the stomach and bowel, and acting upon the heart in a reflex manner. In neurogenic arhythmia, which is especially common in young nervous individuals, the irregularity is often closely related to phases of the respiration, and usually all the beats are of the same strength and volume. It is often abated by atropin.

Myogenic Arhythmia.—Several types are recognized:

(1) *Extra-systoles.*—Extra-systoles are premature cardiac contractions arising from some abnormal site (ventricle, auricle, or, very rarely, auriculo-ventricular node), in the myocardium and due to increased myocardial excitability. The extra beat occurs from time to time following a normal beat at a shorter interval than the space between two of the normal beats. The interval following the extra-systole is longer than the average, and the succeeding regular beat is unduly strong and often manifest to the patient as a precordial thump. Irregularity in rhythm of the heart from this cause is very common.

Intermission of the pulse occurs when the refractory period (the period in which the ventricle is unresponsive to stimuli), following an extra-systole, overlaps the time of the next physiologic stimulation. True intermission must be distinguished from simulated intermission in which the ventricular contractions occur, but are too feeble at times to produce a peripheral pulse. A bigeminal pulse occurs when each normal systole is regularly followed by an extra-systole and a compensatory pause, and a trigeminal pulse occurs when a normal systole is regularly followed by two extra-systoles and a compensatory pause.

In rare instances the stimuli producing the extra-systoles arise in the auriculo-ventricular bundle (true nodal rhythm) causing the auricles and ventricles to contract almost simultaneously, as shown by the superposition of the auricular and ventricular waves (*a* and *c* waves) of the jugular pulse.

In many instances extra-systoles are not due to organic disease of the heart, but are caused by increased functional irritability of the myocardium, by the action of toxic agents (tobacco, tea, etc.), or by conditions acting reflexly on the heart.

(2) *Auricular Fibrillation.*—In this condition the uniform contractions of the auricle as a whole are replaced by a multitude of haphazard fibrillary contractions, and as a result the ventricular beats, and hence the pulse beats, become grossly irregular, both as to time and to force. The pulse rate is, as a rule, increased (110–150), but it may be nearly normal, or if heart-block coexists, even decreased. Other important signs are the absence of normal auricular waves in tracings taken from the jugular vein and from the cardiac apex, and in cases of mitral stenosis, the replacement of the rumbling presystolic murmur by a soft diastolic murmur. Symptoms of cardiac insufficiency are generally present, although some patients enjoy fair health for months or even years. Auricular fibrillation is the result of inflammatory or degenerative changes in the myocardium, and is especially common in cases of mitral stenosis. Once established, it is usually permanent, although in some instances it is paroxysmal. It occurs in more than 70 per cent. of all cases of heart failure with dropsy (Mackenzie).

(3) *Heart-block.*—This form of arhythmia is due to the failure of the auricular contractions to reach the ventricle, owing to defective conductivity in the bundle of His. In complete heart-block the conducting function of the bundle is entirely lost and the ventricle develops an independent rhythm of its own, the pulse being usually regular and numbering about 30 per minute. In partial heart-block the conductivity of the bundle is merely impaired, the result being a prolongation of the interval between the auricular and the ventricular contractions (*a–c* interval) or the dropping of

one ventricular beat in 6, 5, 4, etc. The diagnosis can always be made with instrumental aid and often without it; the association of bradycardia with a normal number of auricular contractions, as shown by inspection of the jugular vein, being characteristic. Heart-block may result from organic lesions in the conducting bundle, such as fibrosis, gumma, abscess, etc., from infectious toxemias or from the action of certain drugs, especially digitalis.

In some cases of complete heart-block (occasionally even in the absence of it) the *Adams-Stokes syndrome* develops. This is characterized by a very infrequent pulse (5 to 40 per minute) and recurring attacks of a syncopal, epileptiform, or vertiginous character, probably the result of cerebral anemia. In the aged the Adams-Stokes syndrome is almost invariably an expression of degenerative myocarditis; in young adults it is usually indicative of syphilitic myocarditis, although in rare instances it may be of nervous origin.

(4) *Pulsus Alternans.*—In this condition there is a diminution in the contractile power of the heart resulting in the occurrence of alternating large and small beats. The pulse may be regular, but as a rule the small wave is delayed, so that the interval between the large beat and the small beat is greater than that between the small beat and the next large one. With extrasystoles the reverse occurs, that is, the small beat is premature. Pulsus alternans if persistent is of evil import.

(5) *Pulsus Paradoxus.*—The characteristic feature of this pulse is a marked diminution in the size of the beats or an actual suppression of beats during full inspiration. It is probably due to some hindrance to the inflow or outflow of blood from the heart, caused by traction on the great vessels. It is not infrequent in adherent pericardium and disease of the mediastinum.

In many instances the type of cardiac irregularity present can be recognized by the palpating finger and the stethoscope, but very often the actual condition can be determined only with the aid of the polygraph or the electrocardiograph.

The Dicrotic Pulse.—This is a pulse in which the main beat is quickly followed by a secondary wave or slight rebound of the vessel. It is frequently observed when the tension is low and the arteries are relaxed, as in exhausting fevers, like typhoid.

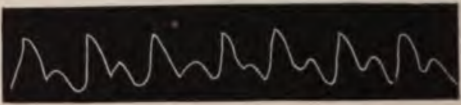

FIG. 7.—Sphygmogram of a dicrotic pulse.

Venous Pulse.—An abnormal (systolic) jugular pulse-wave is seen in tricuspid regurgitation. A venous pulse in the dorsum of the hand may be due to forcible propulsion of the blood through the capillaries, as in compensated aortic regurgitation, or to extreme relaxation of the arterioles, permitting the transmission of the pulse-wave, as in grave anemia.

Capillary Pulse.—This may be detected by the occurrence of systolic blushing in an area of the skin (preferably over the forehead) made hyperemic by friction, or in the everted lip which has been somewhat blanched by pressure of a glass slide. A capillary pulse is sometimes observed in aortic regurgitation, in severe anemia, in exophthalmic goiter, and in neurasthenia.

Asymmetric radial pulses may result from: (1) Anomalies in the distribution, size, or division of one of the vessels. (2) Aortic aneurysm. (3) An embolus or an atheromatous plate within the vessel. (4) Fractures, luxations, or inflammatory exudations causing compression of the vessel. (5) Compression of one vessel by tumors within or without the thorax.

"Water-hammer Pulse" (Corrigan's Pulse).—This pulse is characterized by a quick, powerful beat, which suddenly collapses or recedes. The peculiar pulsation may be distinctly visible, not only in the carotids, but throughout the brachial artery. This pulse is diagnostic of aortic regurgitation during the period of compensation, and its force is due to the excessive ventricular hypertrophy and to the large amount of blood expelled with each systole; its sudden recession is due to the incompetent valves failing to support the column of blood in the aorta.

Tardy Pulse.—The wave rises gradually, is prolonged, and falls away slowly. In the tracing the ascending and descending limbs of the curve are sloping and the summit is blunt. A pulse of this type occurs in aortic stenosis.

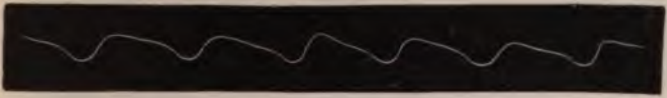

FIG. 8.—Pulsus tardus in aortic stenosis (Strümpell).

The Blood-pressure.—This depends upon: (1) The strength of the heart; (2) the resistance in the vessels and their elasticity; and (3) the volume, and, to a less extent, the viscosity of the blood. Blood-pressure may be roughly estimated by noting the amount of force that is required to arrest the pulse at the wrist when the fingers are pressed upon the radial artery. It can be determined accurately only by means of the sphygmomanometer. The maximal blood-pressure in healthy persons at rest averages about 110 to 135 mm. for adults and about 90 to 110 mm. for children.

Abnormally high blood-pressure or hypertension (150 to 250 mm. or more) may be observed in chronic nephritis, arteriosclerosis, hypertrophy of the heart, angina pectoris, cerebral affections causing increased intracranial pressure, gout, and lead-poisoning.

Abnormally low blood-pressure or hypotension (110 to 80 mm. or lower) is observed in shock and collapse, acute infectious diseases, pulmonary tuberculosis, Addison's disease, many cases of acute and chronic cardiac disease, cachectic states, hemorrhage and conditions causing marked loss of body fluids, and after the administration of certain drugs, such as the nitrites, aconite, etc.

PALPITATION

Definition.—A rapid and tumultuous action of the heart perceptible to the patient. Rapidity not perceptible to the patient is not termed palpitation.

Etiology.—It may result from: (1) Reflex irritation, as from flatulent distention of the stomach. (2) Excitement, mental or physical. (3) Organic heart disease. (4) Exophthalmic goiter. (5) Overwork, as in the "irritable heart" of untrained recruits. (6) Anemia. (7) Neurasthenia or hysteria. (8) Paroxysmal tachycardia.

DROPSY

Definition.—An unnatural collection of serous fluid in the tissues or cavities of the body.

Etiology.—Dropsy may result from: (1) Chronic visceral affections that bring about venous stasis, as chronic heart disease, cirrhosis of the liver, and emphysema. (2) Local obstruction to the venous circulation by emboli, thrombi, tumors, etc. (3) Changes in the composition of the blood, as in anemia. (4) Increased permeability of the capillary walls, as in Bright's disease. (5) Disturbed innervation, as in hysteria, angioneurotic edema, and neuritis.

GENERAL CYANOSIS

Definition.—Blueness of the surface from insufficient oxidation of the blood.

Etiology.—Cyanosis results from: (1) Affections preventing the free entrance of air to the lungs, as in laryngeal or tracheal stenosis, asthma, emphysema, pneumonia; (2) obstruction to the venous return, as in uncompensated cardiac disease; (3) congenital heart disease, in which there is venous congestion or a direct admixture of arterial with venous blood; (4) erythremia (see p. 178); (5) conditions associated with methemoglobinemia, as acetanilid or antipyrin poisoning.

DISEASES OF THE HEART

PERICARDITIS

Etiology.—Inflammation of the pericardium is invariably a secondary process. (1) It may occur in infections generally, in particular rheumatism and chorea. (2) It may result from the extension of disease in adjacent structures, such as the lungs, pleuræ, mediastinal glands, ribs, etc. (3) It is not uncommon as a terminal condition in chronic diseases of various kinds, especially chronic nephritis. (4) It rarely follows local traumatism. The microorganisms most commonly concerned are the pyogenic cocci, the pneumococcus, and the tubercle bacillus.

Pathology.—The membrane becomes red, sticky and lusterless and in a short time a serofibrinous, fibrinous, or purulent exudation makes its appearance. In *serofibrinous pericarditis* there is, as a rule, only a slight fibrinous deposit, the exudation being composed chiefly of straw-colored fluid (from a few ounces to 2 pints or more). Occasionally the fluid is hemorrhagic, particularly in tuberculous cases. In the milder attacks the effusion may be completely absorbed, but in many cases adhesions form here and there between the surfaces and the pericardium becomes permanently thickened.

In *fibrinous pericarditis* little fluid is effused, but the heart is covered with a butter-like exudation, which ultimately undergoes organization and leads to partial or complete obliteration of the pericardial sac. Associated with obliterative pericarditis there are frequently adhesions between the external surface of the pericardium and surrounding parts, and also an increase of the fibrous tissue in the mediastinum (*indurative *mediastinopericarditis.* In other cases the pleuræ and the peritoneum share in the chronic inflammatory process, producing the condition described as *multiple serositis* (Pick's disease). Pericardial adhesions offer resistance to the ventricular contractions and ultimately cause hypertrophy and dilatation of the heart.

In *purulent pericarditis* from a few ounces to 2 or 3 pints of pus may be present in the sac. In rare instances recovery may occur, even without the evacuation of the pus, the latter becoming inspissated and finally infiltrated with lime salts.

In all forms of pericarditis the lesions extend, as a rule, a variable distance into the myocardium.

Symptoms.—Precordial pain or discomfort, palpitation, dyspnea, moderate fever, and weakness are the usual symptoms, but in many cases the disease is latent and only discovered on routine examination. The face may be unduly pale or distinctly cyanosed, the veins of the neck may be turgid, and occasionally, if the effusion is large, there may be hoarseness from pressure upon the recurrent laryngeal nerve or difficulty in swallowing from pressure on the esophagus.

PHYSICAL SIGNS.—In the early stage of serofibrinous pericarditis and throughout in fibrinous pericarditis the only characteristic sign is the friction sound. This is a superficial rubbing or creaking sound, usually double (to-and-fro) and of cardiac rhythm, but not absolutely synchronous with the normal cardiac sounds. It is best heard, as a rule, in the fourth left intercostal space and not transmitted beyond the precordium. Pericarditis with effusion is manifested by several definite signs:

Inspection.—The precordium may be abnormally prominent, especially in children.

Palpation.—The apex-beat is feeble or lost. A pulsation is sometimes apparent in the fourth interspace.

Percussion.—The area of cardiac dulness is increased and irregularly pear-shaped with the base directed downward and the stem upward. It often extends above the third costal cartilage, a considerable distance to the left of the cardiac impulse, and into the right fifth intercostal space an inch or more to the right of the sternum (Rotch's sign), sometimes changing the cardio-hepatic angle, normally about 90 degrees, to an obtuse angle (Ebstein's sign). Other signs of variable constancy include change in the outline of dulness with change of the patient's position, unusual prominence of the sternal end of the first rib (Ewart), and an area of dulness with bronchial breathing near the angle of the left scapula (Bamberger's sign).

Auscultation.—The heart-sounds are indistinct (muffled) and often disproportionately feeble in comparison with the strength of the pulse. The friction sound sometimes persists.

Purulent Pericarditis.—The symptoms are similar to those of serofibrinous pericarditis, but septic phenomena—irregular fever, chills, sweats, pallor, and marked leucocytosis—are often present, and occasionally the precordial tissues are edematous. In doubtful cases recourse should be had to exploratory puncture.

Adherent Pericardium.—The diagnosis cannot always be made during life, but the following signs are suggestive. Enlargement of the heart with (1) systolic retraction in the region of the apex and also posteriorly in the region of the eleventh and twelfth ribs (Broadbent's sign); (2) inspiratory swelling of the cervical veins (Kussmaul's sign); (3) diastolic collapse of the cervical veins (Friedreich's sign); (4) absence of shifting of the apex-beat with change of the patient's posture and during full inspiration; (5) deficiency in the respiratory movements of the diaphragm; (6) the pulsus paradoxus (see p. 201). With these physical signs there are often symptoms of cardiac failure —dyspnea, edema of the legs, etc., or evidences of hepatic cirrhosis—ascites, splenic enlargement, etc. (see perihepatitis, p. 108).

Diagnosis.—*Acute Endocarditis.*—Endocardial murmurs have not the peculiar rubbing or creaking quality of friction sounds, are not affected by pressure with the stethoscope, are heard most distinctly at valve points, are often propagated beyond the precordium, and have a definite relation to the sounds of the heart.

Cardiac Hypertrophy.—The enlargement develops slowly; the impulse is powerful; the apex-beat is displaced downward, and the cardiac sounds are loud.

Cardiac Dilatation.—In this condition there is frequently a wavy impulse over the precordium, the apex-beat is often displaced downward; the dulness is more globular in shape and rarely extends to the left beyond the cardiac impulse, upward above the third rib, or far into the right fifth intercostal space; the cardio-hepatic angle remains a right angle; the boundary between the dulness and pulmonary resonance is much less abrupt than in effusion; and, finally, the cardiac sounds, though weak, are usually clear.

Prognosis.—In the dry and serofibrinous forms the prognosis is good under favorable conditions. In the purulent form the outlook is extremely grave. The fibrinous form, though not immediately fatal, is very serious on account of the secondary changes that it induces in the cardiac muscle.

Treatment.—Absolute rest is imperative. The diet should be light, but nutritious. Locally, an ice-bag is serviceable. Leeching is beneficial in robust subjects. Blisters are useful when there is great pain. In rheumatism salicylate treatment should be continued. Opium is often necessary to secure rest and to allay pain. If heart-failure occurs, such stimulants as whisky, strychnin, digitalis, and caffein must be employed.

Pericardial Effusion.—If the effusion is serous, absorption may be aided by the application of small blisters, by the administration of diuretics—digitalis, caffein, theobromin—and by the administration of saline purges. Potassium iodid is of doubtful efficacy. Diaphoretics, particularly pilocarpin, should not be used. If pressure symptoms become urgent or the effusion does not yield after a thorough trial to the measures just mentioned, paracentesis should be performed. The puncture may be made in the left fifth intercostal space about an inch from the sternum, or in the left costosternal angle, the needle being directed upward and backward.

In pericarditis with purulent effusion the indications are to incise the sac and to afford the freest possible outlet for the pus. The mortality is high (about 60 per cent.) because of the associated conditions. In adherent pericardium the treatment is that of chronic valvular disease. In cases with marked systolic retraction of the precordial region resection of the underlying ribs and costal cartilages (cardiolysis) has been done with some success.

OTHER AFFECTIONS OF THE PERICARDIUM

Hydropericardium (dropsy of the pericardium) results from the causes of general dropsy, especially heart disease or nephritis. The physical signs are those of serofibrinous pericarditis in the stage of effusion.

Hemopericardium (blood in the pericardium) may result from traumatism, the rupture of an aneurysm, or the rupture of the heart itself. In cancerous and tuberculous pericarditis the serous exudate is often more or less bloody.

Pneumopericardium (air in the pericardium) is very rare. It may result from trauma or the rupture of an adjacent air-containing organ. Thus it may be produced by pneumopyothorax, a phthisic cavity, or ulceration of the esophagus or stomach. The symptoms are those of pericarditis. The characteristic physical signs are the occurrence of tympanitic and dull areas in the precordial region changing in relation with changes in the patient's posture and a loud churning murmur ("mill-wheel" sound) on auscultation.

ENDOCARDITIS

(Valvulitis)

Definition.—Inflammation of the lining membrane of the heart. The process is usually confined to the valves.

Varieties.—(1) Simple, benign, or verrucose endocarditis. This begins as an acute affection, but usually leads to chronic sclerotic valvular disease. (2) Malignant or ulcerative endocarditis. No sharp line, either clinically or anatomically, can be drawn between this and simple endocarditis. The terms malignant and ulcerative are used to designate a more intense infection. (3) Chronic or sclerotic endocarditis. This may be the continuation of acute endocarditis or it may be chronic from the outset.

Etiology.—*Acute endocarditis* usually results from acute articular rheumatism, chorea, one of the infectious fevers, or septicopyemia. Gonorrhea, arthritic purpura and acute tonsillitis are less common causes. At least 60 per cent. of all cases of acute articular rheumatism are complicated with endocarditis. The young are more liable to be attacked than the aged. Sixty-two of 73 fatal cases of chorea collected by Osler showed endocarditis. In rheumatism and chorea the mitral valve is most frequently affected and the valves of the right heart are seldom involved. Of the infectious fevers, scarlatina and pneumonia are most prone to heart complications.

The *malignant* type is prone to develop on valves already the seat of chronic inflammation, but it may be primary. It usually follows septicopyemia, pneumococcic infection, gonorrhea, or one of the specific fevers. It is not common in rheumatism or chorea.

The microorganisms most frequently detected in the lesions of acute endocarditis are the streptococci and staphylococci ("micrococcus rheumaticus"), the pneumococcus and the gonococcus.

Chronic endocarditis may be congenital, may follow an acute attack, or may develop insidiously as a sequence of syphilis, gout, alcoholism, plumbism, or chronic nephritis. Overuse of the muscles favors its occurrence; and like arteriosclerosis, which it often accompanies, it may be an expression of senility.

Pathology.—Postnatal endocarditis most commonly involves the valves of the left side of the heart. Prenatal endocarditis most commonly involves the valves of the right side of the heart.

In simple endocarditis the surface of the valve becomes red, swollen, and lusterless. Later, a row of bead-like vegetations (thrombi) appears along the line of maximum contact, which is about 2 mm. from the free margin of the valve. Microscopically, the endothelium beneath the vegetations shows evidence of necrosis, and the adjacent tissue, a round-cell infiltration. The vegetations may be whipped off by the blood-current and carried to distant organs, as the brain, kidney, or spleen; but in the vast majority of cases, if life is preserved, they are transformed, together with the cellular exudate, into fibrous tissue (*chronic endocarditis*), which not only thickens the valves, but, by contracting, so shortens and distorts them that they are rendered in one instance obstructive to the onward flow of blood, and in another incompetent to close the orifice over which they preside. Finally, retrograde changes ensue, the thickened valves becoming fatty and calcareous.

The myocardium is probably more or less involved in every case of endocarditis.

The *malignant* type is characterized by luxuriant vegetation and more or less extensive necrosis and ulceration of the underlying tissues. Destruction of valve leaflets, chordæ tendinæ, etc., are not uncommon sequels and in some cases the inflammation spreads to the cardiac walls (mural endocarditis) or to the great vessels. Embolic showers frequently result from disintegration of the vegetations.

Symptoms.—In many cases of *simple acute endocarditis* there are no subjective disturbances and the only evidence of a valvular lesion is an alteration in one of the heart sounds (most commonly the first sound at the apex), which gradually develops into a definite murmur. In some cases, however, an increase in the fever, cardiac overaction out of proportion to the degree of temperature, irregularity of the pulse, precordial distress and dyspnea are also present.

In *malignant endocarditis* the general symptoms may be those of a frank septicopyemia—irregular fever, sweats, chills, progressive anemia and emaciation, leukocytosis, etc.—or the picture may be more like that of typhoid fever or of acute meningitis. Symptoms and physical signs referable to the heart, such as an increased pulse-rate, palpitation, precordial distress, dyspnea, and a murmur at one or other of the valve-points, are usually, but not invariably, present. Marked variations in the intensity and character of the murmurs from day to day are often noted and are significant. Embolic phenomena occur sooner or later in a larger proportion of cases and are of the greatest diagnostic value. Lodging in the skin emboli may cause a petechial eruption (especially significant); in the kidney, lumbar pain and hematuria; in the spleen, painful enlargement of the organ; in the lung, hemoptysis, with localized dulness and fine rales; in the brain, aphasia, hemiplegia or monoplegia; in the eye, retinal hemorrhage or optic neuritis; and in the large vessels of the limbs, gangrene or infective aneurysms. In doubtful cases blood cultures may afford valuable diagnostic aid.

Malignant endocarditis usually lasts from two to six or eight weeks, but in some instances the infection is more subdued and continues for many months (*chronic or subacute infective endocarditis*). This form is most commonly associated with the *streptococcus viridans*, and is characterized by remittent or intermittent fever, irregular chills, progressive anemia and, not rarely, by the occurrence of fugitive tender nodules in the subcutaneous tissue.

In *chronic sclerotic endocarditis* the symptoms are entirely due to the mechanical effects caused by leaks or obstruction at the valves and to weakening of heart muscle (see chronic valvular disease, page 213).

Diagnosis.—*Typhoid Fever.*—The gradual onset, the more regular fever, the abdominal symptoms, the roseolar rash, the Widal reaction, the bronchial catarrh, the early enlargement of the spleen, and the absence of leukocytosis and of embolic phenomena will serve to separate typhoid fever from malignant endocarditis.

Malarial Fever.—This may be recognized by the presence of the malarial parasite in the blood and by the absence of leukocytosis and cardiac signs.

Prognosis.—Acute simple endocarditis does not often prove fatal, but it rarely leaves the valve undamaged. Under favorable conditions, however, compensatory hypertrophy of the heart ensues and good health may be preserved for an indefinite period. Rapid dilatation of the heart indicates concurrent myocarditis and is a serious sign. Malignant endocarditis almost always proves fatal, but occasionally recovery ensues with more or less permanent damage to the heart.

Treatment.—The treatment of acute endocarditis is mainly that of the causal condition. *Prolonged and complete rest* is of the greatest importance. The patient should be confined to bed not only during the attack, but for several weeks after it has subsided, in order to allow sufficient time for the damage to be repaired or for compensatory hypertrophy to be established.

Externally, an ice-bag is often useful in allaying excitement of the heart. Mild mercurial or saline aperients may be used from time to time for their depurative effect. Digitalis may be of service if the pulse is weak and irregular, but generally it is not indicated. Heart-failure is to be combated by such stimulants as alcohol, ammonia, strychnin, and caffein. Repeated vesication and the prolonged use of potassium iodid have been warmly advocated.

CHRONIC VALVULAR DISEASE

PERIOD OF COMPENSATION

Compensation is effected by an increase in the strength and size of certain cardiac chambers sufficient to enable the arterial system to receive its normal supply of blood, notwithstanding obstruction or regurgitation at one or more of the valves. The duration of this period is indefinite, and depends largely upon the nature and extent of the valvular defect, the amount of damage sustained by the cardiac muscle, and the hygienic conditions to which the patient is subjected. During the period of compensation the existence of a valvular defect may be revealed only by physical signs, and a lessened response of the heart to demands for increased activity, subjective disturbances being trivial or imperceptible.

Aortic Stenosis.—*Definition.*—Obstruction to the flow of blood into the aorta from thickening or adhesion of the aortic segments. Uncomplicated aortic stenosis is a rare lesion. It occurs usually in elderly persons as a part of general arteriosclerosis.

Physical Signs.—*Inspection.*—In many cases, the apex-beat is forcible and is displaced downward and to the left.

Palpation confirms inspection, and often detects a systolic thrill at the base of the heart.

Percussion usually reveals an increased area of cardiac dulness, especially in the long axis of the heart.

Auscultation.—A harsh systolic murmur is heard at the base of the heart and propagated into the carotids.

Pulse.—The pulse is characteristic. It is small, rises and falls very slowly (pulsus parvus et tardus), and is fairly tense.

Compensation and Sequences.—Interference with the discharge of blood into the aorta is followed by hypertrophy of the left ventricle. Ultimately, when the myocardium becomes enfeebled, dilatation ensues and leads to imperfect closure of the mitral orifice, that is, to relative mitral insufficiency.

Diagnosis.—The harshness of the murmur, weakness of the second sound, palpable thrill, enlargement of the left ventricle, and especially the characteristic pulse will serve to distinguish aortic stenosis from other conditions causing systolic murmurs in the aortic area, such as anemia and atheroma of the aorta.

Aortic Insufficiency.—*Definition.*—Failure of the aortic valves to prevent the return of blood into the left ventricle because of (1) shrinking of the leaflets, the result of endocarditis or sclerosis; (2) rupture of the leaflets from mechanical strain or ulceration (comparatively rare); or (3) enlargement of the aortic ring from dilatation or aneurysm of the aortic arch.

The sclerotic form is the most common. It is especially frequent between the ages of thirty and fifty, following syphilis, alcoholism, or undue muscular exertion.

Physical Signs.—Inspection.—The cardiac impulse is diffuse and forcible and the apex-beat is displaced downward and outward. The peripheral arteries, especially the carotids, often throb violently, and in many subjects there is bulging of the precordium.

A capillary pulse is present in the majority of cases. It is shown in the alternate blushing and paling, which occurs when slight pressure is made on a finger nail or a glass slide is pressed upon the lip. Occasionally, pulsation may be observed in the superficial veins on the back of the hand.

Percussion reveals a marked increase in the area of cardiac dulness, especially toward the left and downward.

Auscultation.—There is a diastolic murmur, most distinct at the base of the heart, in the second or third interspace, near the right or left border of the sternum, and transmitted down the sternum. The aortic second sound is usually suppressed. In the majority of cases a systolic murmur is also heard at the aortic area. In some instances a presystolic murmur (Flint murmur) is audible at the apex. It is probably due to the impact of the regurgitating blood stream upon the anterior mitral leaflet. Auscultation over the large arteries, especially the femorals, sometimes reveals a sharp systolic shock ("pistol-shot" sound), and if slight pressure be made with the stethoscope, a double murmur (Duroziez's sign).

The *pulse* is characteristic and is known as the Corrigan or water-hammer pulse. The wave is very large, with a quick upstroke and sudden fall. It is best perceived by elevating the arm and grasping the forearm above the wrist. The sudden collapse of the pulse, as well as the pulsation in the capillaries and superficial veins, is probably due not so much to the prompt return of a large volume of blood into the ventricle as to the reflex dilatation of the arterioles caused by an increase in the intraventricular pressure.

Compensation and Sequences.—The increased pressure within the left ventricle during diastole results in marked dilatation and hypertrophy of that chamber, the heart in consequence becoming enormously enlarged (*cor bovinum* or ox-heart). Ultimately, relative mitral insufficiency ensues from stretching of the mitral ring.

Mitral Stenosis.—*Definition.*—Obstruction to the flow of blood through the mitral orifice, the result of thickening or fusion of the valve-leaflets. Mitral insufficiency often coexists. The disease may follow acute endocarditis or develop gradually in association with arteriosclerosis. The endocarditic form, the more common form, is observed chiefly in the young.

Physical Signs.—*Inspection.*—The apex-beat is, as a rule, not much displaced. Some bulging is often noticed over the lower part of the sternum.

Palpation.—In the majority of cases a very characteristic purring thrill is to be felt in the region of the apex. It is diastolic or presystolic in time, sharply localized, and ends abruptly with the apex-beat.

Percussion usually reveals an increase in the cardiac dulness chiefly to the right of the sternum.

Auscultation.—In compensated cases a rumbling or vibratory murmur is heard a little to the right of the apex late in diastole (presystolic) or throughout diastole. This murmur is confined, as a rule, to a small area, becomes louder toward the close of the diastole (crescendo-like), and ends abruptly in a loud, snapping first sound. It disappears when auricular fibrillation occurs. The second pulmonic sound is usually accentuated. The *pulse* is in no way characteristic. It becomes very irregular, however, with the occurrence of auricular fibrillation (see p. 200).

Compensation and Sequences.—The first strain is upon the left auricle, which becomes enormously enlarged. With overdistention of the left auricle the intrapulmonary pressure is increased and this results in hypertrophy or dilatation of the right ventricle. Ultimately, relative tricuspid insufficiency is likely to ensue from stretching of the tricuspid ring. Unless mitral insufficiency is also present the left ventricle is not usually enlarged.

Mitral Insufficiency.—*Definition.*—Defective closure of the mitral valve owing to enlargement of the mitral ring, the result of weakening or dilatation of the left ventricle (relative insufficiency), or to contraction and curling of the mitral leaflets, the result of endocarditis or slowly progressing sclerosis. Mitral insufficiency is the most common of the valvular defects.

Physical Signs.—Inspection.—The apex-beat is displaced outward rather than downward. Bulging of the precordium and a diffuse pulsation are often noted.

Percussion.—The area of cardiac dulness is increased transversely, both to the left and to the right.

Auscultation reveals a systolic murmur, loudest near the apex, and transmitted to the axilla and sometimes to the angle of the scapula. The pulmonic second sound is often accentuated.

The *pulse* shows no characteristic changes.

Compensation and Sequences.—Enlargement of the left auricle, increase in the intrapulmonary pressure, and hypertrophy and dilatation of the right ventricle are brought about in the same way as in mitral stenosis. Hypertrophy and dilatation of the left ventricle also occur in response to the large amount of blood discharged into this chamber from the auricle during each diastole. With weakening of the right ventricle relative tricuspid insufficiency is likely to ensue.

Tricuspid Stenosis.—This lesion is rare and frequently mistaken for mitral stenosis, with which it is usually associated. The area of cardiac dulness is increased to the right, and there is a presystolic murmur of maximum intensity over the lower end of the sternum. Marked cyanosis is usually present.

Tricuspid Insufficiency.—The organic form, the result of inflammation or degeneration of the tricuspid leaflets is comparatively rare, but the relative form due to enfeeblement or dilatation of the right ventricle, the result of obstruction in the pulmonary circulation (mitral lesions, emphysema, etc.), primary myocardial disease, or general malnutrition is common. Important signs are cyanosis; distention of the superficial veins; a pulsation in the jugular veins, synchronous with the heart-beat; systolic expansile pulsation of the liver; an increase in the area of cardiac dulness particularly to the right of the sternum; and a blowing systolic murmur of maximum intensity over or near the lower third of the sternum.

Pulmonary Stenosis and Pulmonary Insufficiency.—These lesions are very rare. *Pulmonary stenosis* is usually congenital. Important signs are enlargement of the heart to the right; a systolic thrill in the second left interspace near the sternum; a systolic murmur in the same area, which in contrast to the murmur of aortic stenosis, is not transmitted into the carotid arteries; and suppression or extreme weakness of the pulmonic second sound. In the congenital cases cyanosis is usually present from birth.

The physical signs of *pulmonary insufficiency* resemble those of aortic insufficiency. In the former, however, the heart is enlarged to the right, the aortic second sound is usually distinct and the Corrigan pulse is absent.

PERIOD OF DECOMPENSATION

Decompensation may result from: (1) Increasing damage to the valves or heart muscle, as in lesions of a degenerative type; (2) senility, leading to arterial and cardiac degeneration; (3) intercurrent disease (influenza, pneumonia, acute nephritis, etc.), throwing additional strain upon the heart; (4) undue physical exertion; (5) pregnancy and parturition; (6) mental shock.

Symptoms.—The symptoms are varied and in general are due to venous stasis, local anemia, accumulation of carbon dioxid, toxemias from associated renal or hepatic disease, or gross secondary lesions, such as hydrothorax, ascites, or infarct of the lungs. Dyspnea is the most frequent symptom.

It may be constant or only present on exertion. In many cases it is worse during the night. Asthmatic attacks and Cheyne-Stokes breathing may also occur. Cyanosis is sometimes a conspicuous feature. Cough and expectoration are not uncommon, and as a result of intense pulmonary engorgement or of pulmonary infarct hemoptysis may occur. Edema appears at some period in most cases. It usually begins in the feet and spreads upward, often involving the serous sacs. The right pleura is more often affected than the left.

Gastrointestinal disturbances are frequently present and with failure of the right heart the liver becomes enlarged and tender, and slight jaundice not rarely develops. The urine is usually scanty and albuminous, and may contain erythrocytes and tube casts. Many patients complain of palpitation and of precordial discomfort or pain. True angina pectoris may occur. Cerebral disturbances, such as headache, vertigo, faintness, insomnia, delusions and hallucinations are not infrequent. The pulse is weak and irregularities due to extra-systoles, auricular fibrillation, or heart-block (see p. 199) are very often noted.

In *mitral cases* the pulmonary features (cough, expectoration, hemoptysis, etc.), cyanosis and edema are usually the dominant symptoms. In *aortic* cases precordial pain, pallor (aortic facies), and cerebral disturbances are especially common. Edema is rarely pronounced and embolic phenomena are uncommon. In tricuspid insufficiency orthopnea, gastric disturbances, painful swelling of the liver and ascites are usually conspicuous features.

Prognosis of Chronic Valvular Affections.—The character and intensity of the murmur are of little moment in determining the gravity of the lesion. Much depends upon the nature, site and grade of the valvular defect, the state of the myocardium, the general health of the patient and his age, habits and social condition. The relative gravity of the various lesions, in order from the most to the least serious, is probably as follows: Tricuspid insufficiency, aortic insufficiency, mitral stenosis, aortic stenosis, mitral insufficiency. With a stationary lesion and ample compensation, mitral insufficiency is not incompatible with a long life free from discomfort. Sudden death occurs most frequently in aortic regurgitation. Unfavorable factors, generally speaking, are early childhood, marked enlargement of the heart, dyspnea upon slight exertion, irregular rhythm, the likelihood of a recurrence of the causal factor, lesions in the arteries or other viscera, poor general health and bad habits.

Treatment.—If compensation is well maintained, the treatment should be purely hygienic.

Stage of Decompensation.—Rest, both physical and mental, is the most important element in the treatment. The diet should be nutritious, but readily digestible. In some cases it may be well to restrict the diet for a time to milk, giving 3 or 4 ounces every two hours. When dropsy is present it may be desirable to limit the intake of liquids and to withhold salt from the food. The most reliable cardiac stimulant is digitalis (10 to 30 minims of the tincture two or three times a day). It may be given in any form of valvular disease when there are dyspnea, edema, deficient urination, and a frequent, weak, irregular pulse. It is of the greatest service in auricular fibrillation. Strophanthus sometimes succeeds when digitalis fails. Strychnin is the most valuable adjuvant to digitalis, especially if there are degenerative changes in the heart. Mercurial and saline aperients are useful in lowering venous tension, and without their aid digitalis may fail. If the right ventricle is greatly overdistended and cyanosis is marked, venesection to the extent of half a pint or more may prove life-saving. Iron and arsenic are very serviceable when there is anemia. They may sometimes be combined advantageously with digitalis and strychnin, as in the following pill:

℞.	Arseni trioxidi	gr. ⅓
	Massæ ferri carbonatis	gr. xx
	Strychninæ sulphatis	gr. ss
	Pulveris digitalis	gr. x–xx.—M.

Fiant pilulæ No. xx.

SIG.—One pill after meals.

Dropsy.—The most useful measures are hydragogue cathartics (salines in concentrated solution; compound jalap powder, 30 to 40 grains; and elaterium, 1/8 grain); diuretics (digitalis, caffein, theobromin, theocin, vegetable salts of potassium); the application of smooth, firm bandages to the limbs; and the introduction of fine silver cannulæ (Southey's tubes), or incisions behind the ankles. Hydrothorax and ascites call for tapping, especially if the breathing is much embarrassed.

Restlessness and Insomnia.—On the whole, morphin (1/4-1/6 grain) with atropin (1/150-1/120 grain), preferably hypodermically, is the best sedative. Chloralamid, bromides, veronal and trional are worthy of confidence.

Pain.—Temporary oppression is often relieved by warm or cold applications and the administration of Hoffmann's anodyne. Severe continuous pain may yield to leeching or blistering. In anginoid pains nitrites and potassium iodid are often efficacious. For nocturnal distress morphin is especially useful.

Sudden heart-failure must be met by the administration of diffusible stimulants, such as ammonia and alcohol. The intravenous injection of strophanthin (1/120-1/60 grain) often acts very satisfactorily. It should not be employed, however, if digitalis has recently been used, or repeated within forty-eight hours, since serious cumulative effects may ensue. The application of heat to the precordium is useful.

After-treatment.—The important measures are moderation in diet, regular care of the bowels, suitable rest, alternating with mild exercise, warm but not hot saline baths, and mild frictional massage.

ENLARGEMENT OF THE HEART

Varieties.—(1) *Simple Hypertrophy.*—The muscle of the heart is increased in thickness, but the cavities are of normal size.

(2) *Eccentric Hypertrophy* (*Hypertrophy with Dilatation*).—The muscle is thickened and the cavities are increased in size.

(3) *Simple Dilatation.*—The muscle is thinned and the cavities are increased in size.

Etiology.—*Hypertrophy* is usually the result of increased demands upon the functions of the heart. Thus, it may be due to—(1) Valvular disease; (2) abnormal resistance in the peripheral circulation, as in arteriosclerosis and chronic Bright's disease (left ventricle); (3) abnormal resistance in the pulmonary circulation, as in emphysema and cirrhosis of the lung (right ventricle); (4) prolonged exertion, as in athletes; (5) long-continued palpitation or tachycardia, as in exophthalmic goiter or tobacco heart; (6) interference with the ventricular contractions by pericardial adhesions.

Dilatation of the heart results from the same causes. It is more likely to occur than hypertrophy when the demands are sudden and severe, or when they fall upon a heart the walls of which are already degenerated.

Pathology.—In *hypertrophy* the muscle of the heart is firm and of a dark-red color. The normal weight (8 or 9 ounces) may be doubled or trebled. When the left ventricle is chiefly involved, the organ is increased in length. When the right ventricle is chiefly involved, the organ becomes more globular. Microscopically, the fibers are increased in size and in number.

In *dilatation* the heart muscle is softer, more flabby, and often lighter in color from degenerative changes.

Symptoms.—*Hypertrophy.*—Unless the hypertrophy is more than compensatory, no symptoms result. Excessive hypertrophy may give rise to precordial distress and symptoms of cerebral hyperemia—headache, tinnitus aurium, flashes of light, etc.—and the following physical signs: bulging of the precordium; a heavy impulse; displacement of the apex-beat downward and to the left; an increase in the area of cardiac dulness; a loud, booming first sound; accentuation of the aortic second sound or of the pulmonic second sound, according as the hypertrophy involves the left or right ventricle; and a strong, full pulse.

Dilatation also gives signs of cardiac enlargement, but the impulse is feeble or imperceptible, the first sound is short and weak (clicking), the pulse is rapid and feeble, and often irregular or intermittent, and usually there are symptoms of venous congestion—dyspnea, cough, edema, flatulent dyspepsia, and deficient urination. Soft systolic murmurs, the result of relative mitral or tricuspid insufficiency, may be heard.

Treatment.—In *hypertrophy* treatment is rarely called for. Mercurial and saline aperients are useful in lowering arterial tension. Aconite may be used cautiously. The cerebral symptoms sometimes yield to bromids or to vasodilators, like the nitrites.

The treatment of *dilatation* is that of decompensation in valvular disease.

ACUTE MYOCARDITIS

Definition.—Acute inflammation of the heart muscle.

Etiology.—It results from the same causes as acute endocarditis.

Pathology.—It is usually associated with endocarditis or pericarditis. Occasionally the myocardium is the only part of the heart affected. The inflammatory process is always accompanied with more or less parenchymatous or fatty changes in the muscle-fibers. The characteristic feature is the infiltration of the interstitial tissue with round cells.

Symptoms.—The symptoms are often masked by the primary disease. Dyspnea, precordial discomfort, palpitation, pallor, and weakness of the pulse out of proportion to the severity of the general infection are important manifestations. The pulse is usually rapid and irregular, but there may be bradycardia from vagal disturbance or depressed conductivity of the auriculo-ventricular bundle (heart-block). The first sound at the apex is weak and indistinct or replaced by a soft systolic murmur, and the blood-pressure is, as a rule, low. The heart may or may not be dilated.

Treatment.—The treatment is that of acute endocarditis

The most useful stimulants are ammonia, whiskey, camphor, adrenalin, strychnin and caffein. Digitalis, except in very small doses, is contraindicated.

CHRONIC MYOCARDIAL DISEASE

The chief chronic affections of the myocardium are fatty infiltration, fatty degeneration, and fibroid induration (chronic interstitial myocarditis).

Fatty Infiltration.—This consists in an excess of the fat which is normally present in variable amounts beneath the epicardium, especially along the blood-vessels and in the grooves. In advanced cases the fat is also found between the muscle-fibers. The latter may remain normal for a long period, but ultimately, owing to compression, they undergo atrophy and fatty degeneration. Fatty infiltration results from the causes which lead to general obesity.

The *symptoms* are rarely marked unless the muscle-fibers themselves are affected. It may be suspected in obese subjects when there is complaint of dyspnea and palpitation upon slight exertion, and the heart sounds are indistinct and the pulse is small and weak.

In the early stages the *treatment* is that of obesity—regulation of diet, restriction of liquids, graduated exercise, Nauheim baths, etc. In advanced cases the treatment is that of fatty degeneration of the heart.

Fatty Degeneration.—This is a degeneration of the muscle-fibers themselves, with the formation of fat. It is frequently due to local anemia from sclerosis of the coronary arteries; it follows hypertrophy in valvular disease; it is a common result of malnutrition from old age, wasting diseases, or grave anemia; it is associated with parenchymatous degeneration (cloudy swelling) in severe infections; it occurs also in acute mineral poisoning, as by phosphorus, antimony, or arsenic.

The muscle of the heart is pale, soft, and flabby, and feels greasy to the touch. Microscopically, the fibers are found filled with small, dark fat-granules.

Fibroid Induration.—This is usually met with as a replacement fibrosis secondary to sclerosis of the coronary arteries. It may be secondary, however, to chronic endocarditis or pericarditis. The causes in the main are those of general arteriosclerosis—gout, alcoholism, syphilis, plumbism, overeating, chronic nephritis, etc.

Symptoms of Myocardial Incompetence.—The clinical picture presents many variations. Dyspnea, appearing at first only on exertion, is an almost constant symptom. Palpitation or discomfort in the region of the heart frequently accompanies the dyspnea. Transient giddiness or a tendency to syncope is not uncommon. Paroxysms of angina pectoris or of cardiac asthma may occur. Digestion may be good, but not rarely patients complain of epigastric fulness and eructations. Progressive pallor and weakness often develop from increased venous tension and interference with absorption. Attacks of asystole (orthopnea, cyanosis, pulmonary edema and delirium cordis) are sometimes observed, and occasionally the Stokes-Adams syndrome (infrequency of the pulse with epileptiform, syncopal, or vertiginous attacks) is present. In very many cases the symptoms of chronic nephritis form a part of the clinical picture (*cardiorenal group*) the renal condition standing in the relation of either cause or effect to the cardiac inadequacy. Toward the close the breathing may assume a Cheyne-Stokes type. The heart usually presents some degree of enlargement. The aortic second sound is often relatively accentuated. The first sound at the apex may be indistinct or valvular, or replaced by a systolic murmur. The pulse is extremely variable. It may be abnormally frequent or infrequent. In many cases it is weak, but not rarely in the beginning it is of high tension, the manometer registering from 160 to 200 mm. or more. With increasing incompetence, some form of arhythmia (see p. 198) usually develops.

The *history*, *age of the patient*, and *condition of the arteries* must also be considered in making the diagnosis.

Prognosis.—The condition is always grave and carries with it the possibility of sudden death; nevertheless, with care, the patient may live many years.

Treatment.—Laborious work, mental strain, and excitement should be avoided, as far as possible. The diet should be simple and easily digestible. The use of alcohol and tobacco should be forbidden. If the pathologic changes are not far advanced, graduated exercise, coupled with warm saline baths, as in the well-known Nauheim treatment, may be very efficacious. Constipation must be relieved. As to special treatment, strychnin ($1/60$ to $1/30$ grain thrice daily) is often highly serviceable. When there is anemia, iron and arsenic are excellent adjuvants. In premature arteriosclerosis, especially if there is evidence of syphilis, iodids are of signal benefit. For nocturnal dyspnea no drug is so useful as morphin. Nitrites are beneficial in cases with high arterial tension, anginoid pains, or cardiac asthma. Digitalis is serviceable in some cases; it should be given cautiously, however, and in small doses. Venesection sometimes proves advantageous when the arterial tension is high.

ANGINA PECTORIS

(Neuralgia of the Heart; Stenocardia)

Definition.—A symptomatic affection most commonly associated with occlusion of the coronary arteries and degeneration of the myocardium, and characterized by severe paroxysmal pain in the region of the heart and a sense of imminent death.

Etiology.—It usually develops after middle life, and is very much more common in men than in women. The predisposing causes are those of arteriosclerosis. In some instances an hereditary tendency has been noted, and not infrequently the attacks have been preceded by prolonged mental anxiety.

A false form of angina (*pseudo-angina*), rarely, if ever fatal, sometimes occurs in association with hysteria, vasomotor disturbances or the excessive use of tobacco.

Pathology.—Disease of the coronary arteries, sclerotic aortitis and myocardial degeneration are the lesions usually found after death. Occasionally, typical attacks occur in lesions of the aortic valve, especially insufficiency, and in aortic aneurysm.

Symptoms.—The attacks are usually excited by strong emotion, muscular effort, exposure to cold, or flatulent indigestion, and are characterized by intense pain, radiating from the heart to the shoulder and arm (usually the left), a sense of impending death, dyspnea, and a pale, anxious face. The pulse is very variable. The attacks last from a few seconds to several minutes. Death may occur in the first attack, or there may be recurring attacks over a period of many years.

Hysteric Angina.—This neurosis is seen chiefly in women, whereas true angina is rare in women; there is no evidence of organic heart disease; the attack is often nocturnal, and is longer in duration; emotional outbreaks, such as moaning and crying, are common, and other stigmata of hysteria are usually present.

Gastralgia.—The pain is apt to appear when the stomach is empty, and is relieved by stimulating food; it does not radiate to the shoulder and arm; there is no sense of impending death, and no evidence of structural heart disease.

Prognosis.—True angina is always of grave import. Sudden death may occur at any time. In false angina the prognosis is favorable.

Treatment.—The general treatment is that of chronic myocardial disease. In syphilitic cases specific medication is, of course, indicated. Abstinence from alcohol and tobacco should be strictly observed. The most valuable special remedies, in the order of their efficacy, are the nitrites, iodids, theobromin, and arsenic.

The Attack.—No drug is so generally useful as amyl nitrite (3 to 5 minims on a handkerchief). Marked flatulency should be met by the prompt administration of Hoffmann's anodyne or spirit of mint. If the attacks are severe and prolonged morphin (1/3 to 1/4 grain) and atropin (1/120 grain) should be given hypodermically. If these remedies fail, recourse should be had to chloroform inhalations. The application of heat to the precordium is useful. Cardiac depression following the seizures should be combated by strychnin, ammonia, camphor, or ether.

DISEASES OF THE ARTERIES

ARTERIOSCLEROSIS

(Atheroma; Chronic Arteritis)

Definition.—A chronic disease of the arterial walls, characterized by various degenerative changes and more or less fibrous induration.

Etiology.—It is a natural accompaniment of old age. As a presenile condition it often depends upon syphilis, gout, chronic nephritis, alcoholism, chronic lead poisoning or excessive muscular work. A not uncommon cause is long-continued high arterial tension (*hyperpiesis*), the result of over-eating, lack of physical exercise, and excessive mental strain. The disease sometimes follows acute infections, such as typhoid fever, rheumatism, etc.

Pathology.—The arteries are thickened, tortuous, and rigid. The intima of the large vessels often reveals roughened and opaque areas that may be the seat of calcareous deposits. In extreme cases there may be spots of necrotic softening in the subendothelial tissue, forming so-called "atheromatous abscesses."

Microscopic examination shows changes in all three layers. The intima is more or less thickened and often presents areas of fatty degeneration with calcification (atheromatous plaques). The media is the seat of fatty degeneration and calcification (pipe-stem arteries), or of a diffuse or patchy fibrosis with more or less calcification. The adventitia is always more or less thickened and sclerotic.

Symptoms.—These vary with extent and distribution of the sclerosis. If the process is general, it may be recognized by rigidity of the peripheral vessels, a sluggish, high-tension pulse (150–200 mm. Hg.), accentuation of the second aortic sound, enlargement of the left ventricle, increasing pallor, and a gradual loss of physical and mental vigor.

If the *coronary arteries* are especially involved, the symptoms of chronic myocardial disease appear (see p. 225). If the *renal vessels* are especially affected, there may be symptoms of chronic interstitial nephritis (see p. 146). Involvement of the *cerebral arteries* may be indicated by headache, vertigo, insomnia, mental sluggishness, and, perhaps, transient paralysis. Sclerosis of the *mesenteric vessels* may lead to digestive disturbances and occasionally to attacks of abdominal pain (angina abdominis). Sclerosis of the *arteries of the limbs* may be manifested by painful muscular cramps, sudden lameness or "giving way" of the legs during walking (intermittent claudication), neuritic symptoms (numbness, tingling, darting pains, etc.), and, rarely, by red, painful neuralgia (erythromelalgia).

Sequels.—The most important are cerebral hemorrhage or thrombosis, chronic myocardial disease, angina pectoris, interstitial nephritis, aneurysm, and gangrene of the extremities.

Treatment.—Treatment should be directed to the underlying condition. Alcohol should be forbidden. Tobacco should be used in great moderation. Overexertion, both mental and physical, is injurious. Gentle exercise in the open air, however, may be recommended. The quantity as well as the quality of the food demands careful revision. Turkish baths or Nauheim baths may be ordered in suitable cases. Iodids in small doses, over a long period, sometimes have a very beneficial effect. The periodic use of mild mercurial or saline aperients is serviceable. The nitrites are often useful when the blood-pressure becomes too high, but considerable caution must be exercised in their employment as high tension is usually a conservative and compensatory factor. In robust subjects moderate blood-letting sometimes affords much relief.

ANEURYSM OF THE AORTA

Definition.—A tumor-like sac, containing blood and communicating with the canal of the aorta.

Etiology.—The predisposing causes are those of arteriosclerosis—syphilis, alcoholism, gout, excessive muscular work, lead-poisoning, and nephritis. Of these, syphilis is by far the most potent factor. Immoderate physical exertion is the mo[illegible]mon exciting cause. More than 80 per cent. of all cases [illegible]ur in males. It is most frequent between the ages of thirty and fifty.

Pathology.—Aneurysms are divided, according to shape, into *fusiform*, *saccular*, and *cylindric* forms. Rupture of the intima, with the passage of blood between the outer tunics, constitutes a *dissecting aneurysm*. A *false aneurysm* is one in which all the tunics are ruptured and the extravasated blood is circumscribed by the surrounding connective tissue.

The contents of an aneurysm are either fluid or coagulated blood. In saccular aneurysms the wall is often reinforced by fibrin deposited in lamellar form.

The arch of the aorta is the most common seat. About 10 per cent. of aortic aneurysms are abdominal.

THORACIC ANEURYSM

Physical Signs.—*Inspection* may reveal a circumscribed bulging and an abnormal area of pulsation. Dilatation of the superficial veins may also be noted, and in advanced cases the skin over the prominence may be red and glossy. It is particularly important to examine the back as well as the front of the chest.

Palpation.—This may detect an expansile pulsation, a systolic thrill, and a diastolic shock from the recoil of the blood in the sac.

In aneurysm of the transverse arch a downward tug o trachea is sometimes felt when the head is thrown back an cricoid cartilage is grasped between the fingers and thumb (Oliver's sign).

Percussion may reveal an area of dulness to the right or left of the sternum, over the manubrium, or behind in the left interscapular region.

Auscultation.—Unless the sac contains too much fibrin, the ear may detect marked accentuation of the diastolic sound and a systolic murmur or bruit.

Radioscopy may demonstrate a shadow corresponding to the location of the aneurysmal sac.

Pulse.—There may be inequality of the radial pulses, owing to partial blocking of a main arterial branch or to pressure on the innominate or one of the subclavian arteries by the sac itself.

Pressure Effects.—These are especially marked in aneurysms involving the transverse portion of the arch. *Dyspnea* with *stridulous inspiration* may result from pressure on the trachea or a bronchus. *Bloody sputa* ("*weeping*") may occur from the same cause. *Paroxysmal croupy cough* may be excited by pressure on the trachea or recurrent laryngeal nerve. *Hoarseness or aphonia* may also result from pressure on the recurrent laryngeal nerve. *Dysphagia* may result from pressure on the *esophagus*. *Pain* of a boring or lancinating character may arise from pressure on adjacent nerve-trunks or bones. Attacks of *angina pectoris* may occur as a result of the underlying aortitis. *Inequality of the pupils* and *unilateral sweating* may be excited by pressure on the sympathetic. Edema, cyanosis and enlargement of the veins of one or the other arm may arise from pressure on one of the large venous trunks.

Diagnosis.—*Mediastinal tumor* may simulate aneurysm, but in the former the pulsation is not expansile, there is no diastolic shock, the tracheal tug is usually absent, and there may be cachexia, enlargement of superficial glands, and leukocytosis.

Pulsating Empyema.—A left-sided purulent effusion may transmit a cardiac pulsation, but there is no diastolic shock, no thrill, and no murmur. The history, moreover, will usually suggest pleurisy.

Aortic Stenosis.—In this condition there are no evidences of a tumor, no pressure symptoms, and no inequality changes in the radial pulses.

Prognosis.—The outlook is grave. Death usually occurs in from one to three years from rupture, asphyxia, exhaustion, septic inflammation of the lung ("aneurysmal phthisis"), or cerebral embolism. Rupture may take place into the trachea, a bronchus, the pleura, lung, pericardium or esophagus. Occasionally recovery follows from clot-formation.

Treatment.—The treatment commonly employed is a modification of Tufnell's method, and consists in absolute rest in bed for a period of six or eight weeks, a comparatively dry diet, and the administration of potassium iodid (20 to 30 grains thrice daily), and of calcium lactate (15 grains thrice daily). For severe pain the most effective measures are the application of an ice-bag and the administration of nitroglycerin or morphin. When there is marked dyspnea with cyanosis, venesection may afford prompt relief. Attempts have been made to favor coagulation by injecting gelatin (100 c.c. of a 5 per cent. sterilized solution) subcutaneously; but the results have not been decisive. In some instances the introduction of gold wire into the sac and the passage of an electric current through the wire (Moore-Corradi method) give excellent results. The operation is not a dangerous one and often relieves pain and prolongs life. It is only suitable for saccular aneurysms.

ANEURYSM OF THE ABDOMINAL AORTA

Seat.—It is most frequently located near the celiac axis.

Symptoms.—It may be recognized by sharp pain in the back, radiating along the spinal nerves, by a delay in the femoral pulse, by gastro-intestinal symptoms, and by physical signs similar to those of thoracic aneurysm.

Diagnosis.—An *abdominal tumor* may receive a pulsation from the aorta and simulate aneurysm, but in the former the pulsation is not expansile, and is frequently lost when the patient is placed in the knee-breast posture.

Expansile Abdominal Aorta.—This is most frequently seen in women, in whom abdominal aneurysm is rare; the pulsation is often paroxysmal; there is no distinct tumor, and there are no pressure symptoms.

Course and Treatment.—Death usually results from rupture. Occasionally the fatal issue is effected through erosion of the vertebræ and paraplegia, or through embolism of the superior mesenteric artery. The treatment is that of thoracic aneurysm.

DISEASES OF THE RESPIRATORY SYSTEM

THE NOSE

The Sense of Smell.—This is tested by holding odoriferous substances before one nostril at a time while the other is closed. Pungent vapors should be avoided, as the irritation which they excite, and not their odor, may lead to their recognition.

The sense of smell may be impaired or lost (anosmia) in: (1) Lesions of the brain affecting the olfactory tract or bulb (contusions, tumor, meningitis); (2) lesions of the olfactory nerves (rhinitis, neuritis from influenza, etc.); (3) hysteria; (4) paralysis of the trigeminal nerve, causing dryness, and trophic changes in the Schneiderian membrane.

Extreme acuteness of the sense of smell (hyperosmia) and *perversions of the sense of smell* (parosmia) are sometimes observed in hysteria, epileptic aura, and organic disease of the brain.

Epistaxis.—Hemorrhage from the nose occurs under the following conditions: (1) Traumatism; (2) inflammation or ulceration; (3) new growths; (4) high blood-pressure, as in arteriosclerosis, chronic interstitial nephritis, and hypertrophy of the heart; (5) venous stasis from chronic heart or liver disease; (6) blood dyscrasias, as in hemophilia, purpura, scurvy, pernicious anemia, etc.; (7) onset of acute infections, especially typhoid; (8) ascent to high altitudes; (9) vicarious menstruation (rare).

THE LARYNX

Spasm of the laryngeal adductors is characterized by nse dyspnea and occurs in spasmodic croup; in true

In women and children breathing is somewhat more rapid. The ratio between respirations and pulse-beats is as 1 is to 4 or 4.5.

Rapid respiration (*polypnea*) is noted in excitement; in pyrexia; in inflammatory diseases of the lungs; in anemia; in certain affections involving the base of the brain; in poisoning from certain drugs that affect the respiratory center; in hysteria; in painful affections of the chest as pleurodynia and pleurisy.

Infrequent respiration is observed in certain diseases of the brain, as meningitis, tumor, apoplexy; in advanced fatty degeneration of the heart; in certain forms of coma, particularly uremic and diabetic; in poisoning with certain drugs, especially opium; in obstruction to the air-passages, as in asthma and in laryngeal spasm.

Cheyne-Stokes, or Tidal-wave Breathing.—In this type the respirations gradually increase in depth until they reach a climax, then gradually subside, and finally cease entirely for from five to fifty seconds, when they begin again. Consciousness is sometimes lost during the pause. Cheyne-Stokes breathing depends on some disturbance of the respiratory center the exact nature of which is still undetermined. It is usually a forerunner of death, but in some cases of cardiac and renal disease it persists for months.

Its *chief causes* are: (1) Certain cerebral diseases, as apoplexy, meningitis, and tumor. (2) Advanced cardiac disease, especially fatty or fibroid heart. (3) Certain forms of coma, especially that produced by uremia, opium-poisoning, and sunstroke.

COUGH

Cough may be induced by: (1) Most organic diseases of the pharynx, larynx, bronchi, and lungs. (2) Foreign bodies in the air-passages. (3) Certain infections which are associated with catarrh, such as typhoid fever, measles, whoopin cough, and influenza. (4) Inhalation of irritant dusts vapors. (5) Irritation of nerves that are in anatomic relation with the vagus. (6) Hysteria.

Laryngeal Cough.—This cough has a hard, metallic, ringing intonation, and has been termed "croupy." It is observed in laryngitis; in whooping-cough; in tuberculosis and syphilis of the larynx; when a foreign body is lodged in the larynx; when the recurrent laryngeal nerve is irritated by pressure of a tumor or aneurysm; and in hysteria.

Dry Cough.—Cough without expectoration is especially observed in the beginning of inflammatory diseases of the bronchi and lungs; in pleurisy; in most chest diseases of early childhood; and in reflex irritation of the larynx.

Moist or loose cough occurs especially in bronchitis, bronchiectasis, pulmonary edema, pulmonary tuberculosis, in pneumonia after the crisis, and abscess of the lung.

EXPECTORATION

Mucoid sputum is noted especially in the beginning of acute bronchitis; in asthma; in the early stages of pneumonia and tuberculosis; and in pulmonary edema. In edema the sputum is very frothy and watery.

Mucopurulent sputum is observed in acute bronchitis (later stages), chronic bronchitis, bronchiectasis, pneumonia (after the crisis), pulmonary tuberculosis (later stages), pulmonary abscess, and empyema communicating with the lung. In the last two conditions the expectoration may be composed of pure pus.

Hemorrhagic Sputum.—Sputum has a reddish, brownish, or prune-juice color when mixed with blood. In some cases the sputum consists of pure blood (see hemoptysis). Hemorrhagic expectoration occurs in tuberculosis of the lungs, in pneumonia (especially croupous pneumonia), in hemorrhagic infarctions of the lungs, in abscess and gangrene of the lungs, in tumors of the lungs, and, finally, in congestion of the pulmonary circulation. In cancer of the lungs the sputum sometimes resembles red-currant jelly, and in hepatopulmonary abscesses (amebic) the expectoration has been likened to anchovy sauce.

Albuminous Sputum.—The expectoration of large quantities of serous albuminous fluid rarely occurs suddenly after the aspiration of pleuritic effusions. It is sometimes followed by fatal edema of the lungs.

Fetid sputum usually results from bronchiectasis, advanced pulmonary tuberculosis with cavities, gangrene of the lung, and abscess of the lung.

Such sputum, if allowed to stand in a conic glass, settles in three layers: an upper layer of discolored froth, a middle layer of turbid mucus in which are suspended purulent strings, and a bottom layer of decomposed pus.

Nummular sputum is sputum occurring in round, flat, coin-shaped masses, which sink in water. Expectoration of this character is seen in advanced pulmonary tuberculosis and occasionally in bronchiectasis.

The Microscopy of Sputum.—*Elastic fibers* are found in the sputum in pulmonary tuberculosis, abscess, gangrene of the lungs, and in some cases of bronchiectasis.

The Detection of Elastic Fibers.—Place the sputum which has collected during the night in a glass beaker, and add to it an equal volume of a solution of caustic soda (20 grains to the ounce), and boil over a spirit-lamp, stirring it occasionally with a glass rod. As soon as it boils, pour into a conic glass, and add four or five times the amount of cold distilled water. Allow the mixture to stand for two to three hours, and examine the sediment as for tube casts (Fenwick).

Spirals of Mucin.—Tightly coiled spirals of mucin, which probably represent molds of the fine bronchioles, were first pointed out by Curschmann in the sputum of asthma. They have also been observed in the sputum of croupous pneumonia.

Charcot-Leyden Crystals.—These are small transparent octahedral crystals, similar to those found in the blood in leukemia. They are observed especially in the sputum of asthma. They have also been noted in tuberculosis, in fibrinous bronchitis, and in acute bronchitis.

Crystals of Fatty Acids.—These occur as fine needles, singly or in bundles, and are often sharply curved near their extremities. They are observed in the sputum of chronic bronchitis, of abscess, and of gangrene of the lungs.

Crystals of Hematoidin.—These occur as small yellow needles, rhombic plates or tufts, and are found in sputa which contain altered blood. They may be observed in abscess, gangrene, and cancer of the lungs.

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS

Inspection.—Inspection serves to determine the shape of the chest, any unnatural prominence or depression, the amount of expansion, and any inequality of expansion.

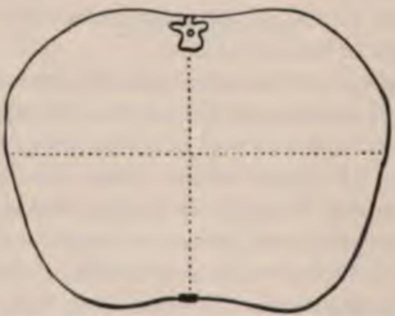

FIG. 9.—An outline of the normal chest.

Phthisinoid Chest.—The anteroposterior diameter is short; the thorax is long and flat; the ribs are oblique; the scapulæ are prominent; the spaces above and below the clavicles are depressed; and the angle formed by the divergence of the costal margins from the sternum is very acute.

Rachitic Chest.—This may resemble the former, but usually the sides are considerably flattened and the sternum prominent, so that the term "pigeon-breast" has been applied to this particular form. The sternal ends of the ribs are enlarged or "beaded," and this characteristic has given rise to the term "rachitic rosary." There is often a circular constriction of the thorax at the level of the xiphoid cartilage (Harison's groove).

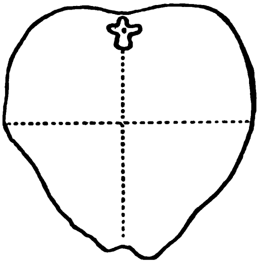

FIG. 10.—Rachitic chest.

Emphysematous Chest.—In advanced emphysema the thorax is short and round; the anteroposterior diameter is often as long as the transverse; the ribs are horizontal; and the angle formed by the divergence of the costal margins from the sternum is very obtuse. The term "barrel-shaped chest" is applied to this configuration.

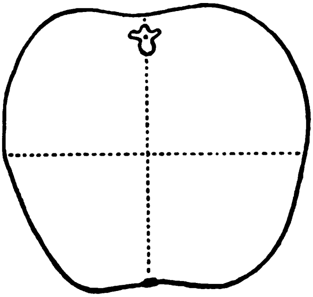

FIG. 11.—Emphysematous chest.

Local Prominences and Depressions.—An unnatural prominence or depression is often observed over the lower part of the sternum, and is usually congenital. The term "funnel breast" or "shoemaker's breast" (because it may result from the pressure of tools) has been applied to this sternal depression.

A unilateral or local depression may be due to: (1) Chronic pulmonary tuberculosis; (2) cirrhosis of the lung; (3) pleurisy with fibrous adhesions.

A unilateral or local prominence may be due to: (1) Pleurisy with effusion; (2) pneumothorax, hydrothorax, hemothorax; (3) an aneurysm or tumor; (4) compensatory emphysema, resulting from impairment of the opposite lung; (5) cardiac enlargements (left side); (6) enlargements of the abdominal organs, especially the liver and spleen.

Expansion.—In women and in children, and in both sexes during sleep, breathing is largely thoracic or costal; in men and in the aged of both sexes it is largely abdominal or diaphragmatic.

Restricted abdominal breathing is observed in pregnancy; in abdominal tumors and effusions; in peritonitis; in diaphragmatic pleurisy; in paralysis of the phrenic nerve from pressure or from bulbar disease; and occasionally in the "hysteric abdomen."

Diminished expansion of one side is observed in pleural effusions; in acute pleurisy (from pain); in consolidation of the lung from tuberculosis, pneumonia, or tumor; in occlusion of a bronchus; and in marked enlargement of the liver or spleen.

Increased expansion of one side is observed in compensatory emphysema.

Litten's Diaphragm Phenomenon.—If a healthy individual is placed in a horizontal position with the feet toward the window and all cross-lights are excluded, a narrow shadow may be seen descending between the sixth and the ninth ribs in each axilla during full inspiration. It is due to the separation of the diaphragmatic pleura from the costal pleura during the inspiratory descent of the diaphragm. This shadow is absent in pleural effusion, pneumonia of the lower lobe, well-developed emphysema, and extensive pleuritic adhesions. As it is present in enlargement of the liver and in subphrenic abscess, it often aids materially in determining whether disease is above or below the diaphragm.

Palpation.—Palpation serves to detect any thoracic tenderness, edema, friction fremitus, or râles, and to determine the vocal fremitus and amount of expansion.

Thoracic tenderness is observed in pleurisy; in pulmonary tuberculosis and pneumonia (from associated pleurisy); in pleurodynia; in intercostal neuralgia (confined to certain spots); in caries and fracture of the ribs; and in contusion and inflammation of the parietes.

Edema of the chest-walls is recognized by "pitting" when pressure is made with the finger. It may be observed in empyema; in deep-seated abscesses of the parietes; after the application of a blister; and in general dropsy.

Friction Fremitus and Râles.—The friction-rub of pleurisy and harsh, sonorous râles can sometimes be detected by palpation.

Vocal fremitus (*tactile fremitus*) is the purring vibration imparted by the voice to the palpating hand.

In determining the vocal fremitus observe the following precautions: Palpate symmetric parts of the chest; make firm pressure; when comparing, use the same pressure on the two sides; apply the hands as nearly parallel to the ribs as possible; and bear in mind that the fremitus is normally stronger over the right chest than the left.

The fremitus is usually slight in women and in children, and in men with thick chest-walls and a weak voice.

Vocal fremitus is abnormally increased in all consolidations of the lung with unobstructed bronchi. It is usually increased in—(1) Tuberculosis; (2) croupous pneumonia; (3) bronchopneumonia. It may be decreased in any of these conditions, however, if the bronchioles contain exudation.

Vocal fremitus is decreased or absent in—(1) Pleural effusions and pneumothorax; (2) emphysema; (3) pulmonary collapse from an obstructed bronchus; (4) pulmonary edema; (5) morbid growth of the lung (if the bronchi are compressed).

Percussion.—Percussion, through the sounds and resistance elicited, serves to determine the boundaries and physical condition of the underlying organs.

Immediate percussion is performed by striking the chest directly with the fingers. It is not often employed, except over the clavicles, where the bones themselves act as pleximeters.

Mediate percussion is performed by using the fingers of one hand as a plexor and those of the opposite hand as a pleximeter; or by using a piece of ivory, glass, or hard rubber as a pleximeter, and a small hammer as a plexor.

The use of the fingers alone is preferable, for only in this way can resistance be determined.

In percussion the following precautions should be observed: Place the finger that is being used as a pleximeter firmly against the chest, and preferably parallel to the ribs; make the finger that is used as plexor strike the one on the chest perpendicularly; fix the forearm, and use no more force than can be obtained from a gentle swing of the wrist. When possible, percuss all parts of the chest anteriorly and posteriorly; percuss both in inspiration and in expiration. In comparing the two sides, be sure to percuss symmetric parts.

Normal Resonance.—On the right side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward to the upper border of the sixth rib in front, and to a line drawn through the tenth spinous process posteriorly.

On the left side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward, within the midclavicular line to the third rib, outside of the midclavicular line to the tenth rib, and posteriorly to a line drawn through the tenth spinous process.

Traube's Semilunar Space.—This is a tympanitic area at the base of the left chest, bounded above by the lung (sixth rib), on the right by the liver, and on the left by the spleen. It is obliterated in pleural effusion on the left side.

Hyperresonance is observed in the following conditions: (1) Pneumothorax. (2) Cavities—tuberculous or bronchiectatic. (3) Emphysema. (4) Lowered pulmonary tension, as above a pleural effusion or consolidation, and in the initial stage of pneumonia (Skoda's resonance). (5) Flatulent distention of the stomach or colon (frequently observed over the base of the left chest).

Tympanitic resonance is resonance of a hollow, drum-like character, like that normally obtained by percussing the empty stomach or the colon. It is elicited over the chest in pneumothorax and cavity-formation.

The cracked-pot sound, or *bruit de pôt fêlé*, is a modified tympanitic sound, and can be simulated by percussing over the cheek when the mouth is partially open. It may be normally heard over the chest of a crying infant (Walshe). In the adult it usually indicates a cavity that has a free communication with a bronchus. It is best detected by keeping the ear near the open mouth of the patient while percussing.

Dulness or flatness on percussion may be caused by the following conditions: (1) Pleurisy with effusion; (2) consolidation of the lung from tuberculosis or pneumonia; (3) collapse of the lung; (4) congestion and edema of the lung; (5) morbid growths in the lung; (6) enlargement of the liver or spleen (at the bases).

Pitch.—Pitch of the note depends largely upon the volume of air, upon the tension of the walls of the cavity, and upon the size of the opening that communicates with the cavity. The less the air, the greater the tension, and the smaller the opening, the higher will be the pitch of the note. It is obvious, therefore, that conditions that are associated with hyperresonance may yield either a high- or a low-pitched note. In beginning tuberculous consolidation the note over the affected apex is higher pitched; but it must be borne in mind that normally the note over the right apex is higher pitched than that over the left.

Resistance.—The sense of resistance appreciated by the percussing finger is increased in proportion as the air in the lungs is decreased. It is generally more marked over a pleural effusion than over a consolidation with patulous bronchi.

Auscultation.—Auscultation of the lungs is practised to determine the character of the respiratory and voice sounds, and to detect adventitious sounds like râles.

In *immediate auscultation* the ear is placed directly over the chest, only a soft towel intervening.

In *mediate auscultation* the sounds are transmitted through a stethoscope, which should be applied to the bare chest.

In auscultation observe the following precautions: Do not exert much pressure with the stethoscope. If the chest is covered with hair, this should be moistened, otherwise it is likely to produce crackling sounds resembling râles. If possible, examine carefully all parts of the chest, anteriorly and posteriorly, during quiet breathing, during full inspiration, during full expiration, and after coughing. Compare carefully the sounds elicited over symmetric parts of the chest.

Normal Respiration.—Vesicular breathing is heard over the body of the lungs and is characterized by a soft, breezy inspiratory sound, and a shorter, lower pitched, less intense expiratory sound. Normally, the expiratory sound is not more than one-third the length of the inspiratory sound. Not infrequently expiration is wholly inaudible. Over the trachea and main bronchi the sounds are harsh and blowing, and the expiratory sound is as long as the inspiratory sound or slightly longer (bronchial breathing).

Modifications of the Respiratory Murmur.—Puerile Breathing and Exaggerated Breathing.—Normal breathing in children is called puerile breathing. Both inspiration and expiration are proportionately increased in length and loudness. Exaggerated breathing has the same characteristics as puerile breathing, and is heard after exertion and over the whole of one lung (compensatory emphysema) if the other is disabled.

Bronchial or Tubular Breathing.—This is harsh, blowing breathing with prolonged, accentuated expiration. The expiration may be considerably longer than the inspiration, and there is often a distinct pause between the two sounds. Bronchial breathing is heard normally over the trachea and in the interscapular space over the large bronchi, and abnormally over consolidated lung, if the bronchi are unobstructed. Thus, it may be heard in croupous pneumonia, in bronchopneumonia, in tuberculosis of the lung, and over the lung above a pleural effusion.

Bronchovesicular Breathing.—This is breathing that is neither bronchial nor vesicular. It is somewhat harsh, and expiration is slightly prolonged and accentuated. Unlike puerile or exaggerated breathing, the normal ratio of inspiration to expiration is not maintained—the expiration is not relatively but *actually* prolonged. Bronchovesicular breathing may be heard when the lung is slightly solidified, as in beginning tuberculosis.

Cavernous or Amphoric Breathing.—This resembles bronchial breathing, but the sounds have a hollow character, and the pitch of the expiration is lower than that of inspiration. It may be imitated by blowing over the mouth of an empty jar.

Cavernous breathing may be heard in the following conditions: (1) Tuberculous or bronchiectatic cavities; (2) pneumothorax, if the opening in the lung is patulous; (3) areas of consolidation near a large bronchus; (4) sometimes over lung compressed by a moderate effusion.

The Breathing of Emphysema.—This is weak breathing, with prolonged low-pitched or inaudible expiration.

Cogged-wheel or Jerky Breathing.—The respiratory murmur is not continuous, but is broken into waves. It is not indicative of any special disease, but it is frequently observed in hysteria, pleurodynia, bronchitis, and incipient tuberculosis.

Weak or Shallow Breathing.—This is noted: (1) When the chest-walls are thick; (2) in the old and feeble; (3) in emphysema; (4) in pleural effusion; (5) sometimes in incipient tuberculosis; (6) in painful affections of the chest, like pleurodynia and beginning pleurisy; (7) in pulmonary edema.

Vocal Resonance.—This is the confused humming sound heard over the chest when the patient speaks. It is modified by the same conditions that modify the vocal fremitus (see page 243).

Bronchophony.—This is exaggerated vocal resonance. It is heard normally over the trachea, and abnormally over consolidated lung (tuberculosis and pneumonia) if the bronchi are unobstructed, over lung that is compressed by pleural effusion, and over some cavities.

Pectoriloquy.—This is a modification of vocal resonance in which the articulate speech is heard very distinctly, as though coming directly from the chest into the ear. It is more pronounced when the patient whispers.

Pectoriloquy is heard over: (1) Cavities that communicate with a bronchus; (2) areas of consolidation in the neighborhood of a large bronchus; (3) pneumothorax, if the opening in the lung is patulous; (4) some pleural effusions.

Egophony.—This is a modification of bronchophony, in which the sounds have a trembling or bleating quality. It is usually heard over slight pleural effusions near the upper border of dulness, especially near the inferior angle of the scapula.

Adventitious Sounds.—These are not modifications of preexisting sounds, but wholly new sounds produced in the lung or pleura. They include râles, friction-sounds, metallic tinkling, and succussion-splash.

Râles.—These are new sounds created in the trachea, bronchi, air-vesicles, or in cavities. They may be due to the passage of air through secretions or other fluid or to the tearing apart of agglutinated alveolar walls by the inspiratory air.

- Pulmonary râles
  - Bronchial
    - Dry
      - Sibilant
      - Sonorous
    - Moist
      - Subcrepitant
      - Bubbling
      - Gurgling
  - Vesicular = Crepitant

Dry râles are probably produced by the passage of air over threads of viscid secretion drawn across the bronchial lumen. They have a musical character and are often transmitted to a considerable distance from the point of production. They are heard particularly in bronchitis and asthma. Sibilant râles are whistling and high pitched; sonorous râles have a humming quality and are lower pitched. Dry râles may be heard on inspiration, expiration, or on both.

Moist râles are probably produced by the rupture of films or membranes of thin secretion stretched across the bronchial lumen. The thinner the liquid and the larger the tube, the coarser will be the râles. They may be heard on inspiration, expiration, or on both.

Subcrepitant or *crackling râles* are fine moist râles. They are heard in all conditions that are associated with liquid in the smaller tubes, such as bronchitis, capillary bronchitis, pulmonary edema, and beginning tuberculosis.

Bubbling râles are coarser than subcrepitant, and are heard in bronchitis, in resolving croupous pneumonia, over softening tuberculous deposits, and over small cavities.

Gurgling râles are very coarse, and resemble the bursting of large bubbles. They are heard over large cavities that contain fluid, and over the trachea in the so-called "death-rattle."

Crepitant Râles.—These are very fine râles, usually heard at the end of full inspiration. They may be simulated by rubbing a lock of hair between the fingers. They have been especially associated with the first stage of croupous pneumonia, and have been ascribed to the forcible separation of adherent vesicular walls. Râles very similar to, if not identical with, these are heard in pulmonary edema.

Friction-sounds are produced by the rubbing together of roughened pleural surfaces. They may be heard both in inspiration and in expiration, and often resemble subcrepitant râles, but they are more superficial and localized than the latter, and are not modified by cough or deep inspiration.

A roughened pleura in the neighborhood of the heart may produce a friction-sound of cardiac rhythm, and one which will still continue when the breath is held; under other conditions pleural friction-sounds cease when respiration is suspended.

Metallic Tinkling.—This name is applied to silvery or bell-like sounds that are heard at intervals over a pneumohydrothorax or large cavity. Speaking, coughing, and deep breathing usually induce them. Care must be taken not to confound them with similar sounds produced by the presence of liquid in a distended stomach.

Succussion-splash or Hippocratic Succussion.—This is a splashing sound produced by the presence of air and liquid in the chest. It may be elicited by gently shaking the patient while auscultating. It is usually associated with hydropneumothorax or a pyopneumothorax.

A similar splashing sound is often heard over a dilated stomach.

Mensuration.—In measuring the sides of the chest observe the following precautions: Measure from the middle of the sternum to the spinous processes; measure both sides after inspiration and after expiration; apply the tape with equal firmness to the two sides. In comparing, measure corresponding levels, and remember that the right side is from half an inch to an inch greater in circumference than the left.

The conditions that render one side more prominent than the other have already been considered.

Radioscopy.—In certain conditions of the chest radioscopy, especially the fluoroscopic screen, furnishes valuable information. It has been found of most service in detecting *aneurysms* and in determining the outlines of the heart in *emphysema.* Some observers have found it useful in detecting *tuberculous consolidation* before any other signs could be elicited. In *pleural effusion* the affected side is dark, the displacement of the heart is well shown, and the diaphragm appears flattened and depressed.

DISEASES OF THE NOSE AND LARYNX

ACUTE CORYZA

(Acute Rhinitis; Cold in the Head)

Acute coryza is an infection of the nasal cavities, sometimes extending into the pharynx, upper respiratory tract, Eustachian tubes and accessory nasal sinuses. Exposure to cold and wet may act as a predisposing factor, but the exciting cause is some bacterium, usually the micrococcus catarrhalis, but sometimes the pneumococcus, or the bacillus of Friedländer. In some cases coryza is symptomatic of a general infection, such as measles or influenza, or of a drug intoxication, such as iodism, or results from the inhalation of irritant dust or vapors.

Symptoms.—The disease is ushered in with chilliness, muscular soreness, general malaise, fulness in the head, and sneezing. The nasal chambers are obstructed, so that the patient is obliged to breathe through his mouth. At first there is no secretion, but in twenty-four or forty-eight hours a watery discharge is established, which later becomes mucopurulent. Slight fever and its associated symptoms are commonly present. The duration is from a few days to two weeks.

Complications.—Extensions of the disease to the frontal sinuses, Eustachian tubes, pharynx, larynx, and bronchi are not uncommon. Repeated attacks may lead to chronic rhinitis.

Treatment.—If the patient is seen at the outset and is willing to remain indoors for twenty-four hours, a hot foot-bath, with a full dose of Dover's powder, followed in the morning by a Seidlitz powder or other saline aperient, often gives excellent results. When the patient must be up and about, the following capsules will usually afford considerable relief:

℞.	Codeinæ sulphatis	gr. iiss
	Pulveris camphoræ	gr. xx
	Ammonii carbonatis	ʒi
	Quininæ bisulphatis	gr. xx
	Extracti belladonnæ	gr. j.—M.

Pone in capsulas No. xx.
Sig.—One every two or three hours.

Warm Dobell's solution (see p. 38) or warm distilled extract of witch-hazel (diluted with 1 part of water) used as a spray at intervals, and followed in a few minutes by an oily application like the following, usually renders satisfactory service:

℞.	Mentholis	gr. iij
	Olei pini pumilionis	♏ v
	Petrolati liquidi q. s. ad	f℥j.—M.

CHRONIC NASAL CATARRH

(Chronic Rhinitis)

Definition.—A chronic inflammation of the nasal mucous membrane.

Etiology.—Repeated attacks of acute coryza, impure air, the continual inhalation of irritating dusts or vapors, lowered vitality, and congenital or acquired obstruction of the nasal chambers are causal factors. It is sometimes an expression of syphilis.

Varieties.—(1) Simple chronic rhinitis; (2) hypertrophic rhinitis; (3) atrophic rhinitis.

Symptoms.—These consist of a mucoid or mucopurulent discharge from the nose; obstruction of the nostrils from swelling or hypertrophy of the mucosa or from inspissated secretion; mouth-breathing; a nasal intonation of the voice; frontal headache; and impairment of the sense of smell.

Symptoms of catarrh of the neighboring organs are frequently present. The most common of these are: dryness of the throat and hawking from pharyngitis; deafness from catarrh of the middle ear; and watering of the eyes from catarrhal occlusion of the lacrimal canal.

Simple Chronic Rhinitis.—The mucous membrane of the nose is congested, swollen, and highly irritable. There is hypersecretion of mucus or mucopus.

Hypertrophic Rhinitis.—The mucous membrane is red and the cavities are more or less occluded from hypertrophy of the cavernous tissue covering the turbinated bones. In advanced cases exostoses from the bony framework are sometimes noted. The secretion is usually composed of thick mucopus. Adenoid growths are often found in the nasopharynx.

Atrophic Rhinitis (*Ozena*).—This form is seen most frequently in young adults, and is more common in women than in men. The nasal chambers are large; the mucous membrane is pale, dry, and glazed; adherent scabs are usually present. The secretion is very abundant, thick, and of a yellowish or greenish color. A characteristic feature is the extremely offensive odor, which is probably due to the decomposition of the retained secretion. In advanced cases there may be necrosis of the bones and sinking in of the bridge of the nose.

Prognosis.—In the simple and hypertrophic form the prognosis is favorable under persistent treatment. In atrophic rhinitis perfect cure is rarely attainable, but great improvement is possible.

Treatment.—Any constitutional vice, if present, should receive appropriate treatment. Fresh air, outdoor exercise, and frequent bathing, with friction of the skin, are to be recommended. Tonics, especially strychnin and cod-liver oil, are often required. The nasopharynx must be kept clean by frequent spraying with an antiseptic alkaline liquid, such as Dobell's solution (see p. 38).

In the hypertrophic form local remedies of an astringent or alterative character are often efficacious. The following are in common use: A mixture of iodin and glycerin containing 6 grains of iodin, 12 grains of potassium iodid, and 1 ounce each of glycerin and water; aqueous solution of ichthyol (20 to 40 per cent.); solution of zinc sulphocarbolate (2 to 5 per cent.); and solution of silver nitrate (1 to 2 per cent.).

If the hypertrophic process prove resistant, the obstruction must be removed by means of caustics (chromic or trichloracetic acid), the galvanocautery, or the snare.

In atrophic rhinitis the crusts may be removed by pledgets of cotton soaked in a solution of hydrogen dioxid. After the nares have been thoroughly cleansed, an oily solution like the following may be applied:

℞.	Mentholis	gr.xx
	Thymolis	gr. vj
	Eucalyptolis	♏xx
	Petrolati liquidi	f℥vj.—M.

For destroying the offensive odor one of the following applications may be used: Pledgets of cotton soaked in an aqueous solution of ichthyol (20 to 50 per cent.); sprays of formalin (1:1000), of Labarraque's solution (1:30), or of potassium permanganate (2 grains to the ounce).

ACUTE CATARRHAL LARYNGITIS

Definition.—An acute catarrhal inflammation of the larynx.

Etiology.—Faulty use of the voice, exposure to cold and wet, the inhalation of irritating dusts or vapors, the use of certain drugs (iodids, mercury), and the lodgment of foreign bodies may cause it. It is also an associated condition in certain infectious diseases, like whooping-cough, measles, diphtheria, and influenza. The organisms most commonly found are the micrococcus catarrhalis pneumococcus, influenza bacillus and streptococcus.

Symptoms.—The chief symptoms are: Hoarseness of the voice or aphonia; hard, ringing cough; pain in the throat, increased by speaking, coughing, and swallowing; expectoration, which is at first scanty and later mucopurulent; slight fever and its associated symptoms. In sensitive persons, and especially in children, paroxysms of croupy cough and dyspnea (*false croup*) may result from spasm of the vocal cords. When there is much edema, severe dyspnea becomes a prominent feature.

Inspection.—The mucous membrane of the laryngeal walls and vocal cords is red and swollen. In grave cases the tissues are highly edematous.

Prognosis.—In simple laryngitis without edema the prognosis is altogether favorable. The attack usually lasts from a week to ten days. When there is edema of the larynx, indicated by dyspnea or threatened asphyxia, the prognosis is grave.

Treatment.—Use of the voice should be avoided. The air of the room should be rendered moist by means of steam. An ice-bag or iodin may be applied externally. At the onset it is advisable to administer a mild aperient. Mild expectorants—ipecac, potassium citrate or ammonium chlorid—may be given in conjunction with heroin, codein, or paregoric if the cough is troublesome. Such a combination as the following is often serviceable:

℞.	Potassii citratis	ʒij
	Vini ipecacuanhæ	fʒiv
	Tincturæ opii camphoratæ	fʒiv
	Syrupi tolutani	f℥iss
	Aquæ	q. s. f℥iv.—M.

SIG.—A dessertspoonful every three hours.

In acute *edematous laryngitis*, if the swelling does not yield to local bloodletting, the external application of ice, astringent sprays (solution of adrenalin chlorid), and scarification of the mucous membrane, tracheotomy should be performed without delay.

SPASMODIC CROUP

(False Croup; Catarrhal Croup)

Definition.—Spasm of the vocal cords, excited by catarrh of the larynx.

Etiology.—The attacks usually occur in young children, and are induced by the causes of catarrhal laryngitis.

Symptoms.—Usually there has been a little hoarseness and cough during the day, and at night the child is awakened from sleep by a severe paroxysm of suffocative cough. The later has a peculiar, hard, metallic quality, and is associated with the evidences of dyspnea, namely: anxious face, dilating nostrils, prominent strenocleidomastoids, and retraction of the base of the chest with each inspiratory effort. During the paroxysm the skin is hot and the pulse is tense and rapid. In from a few moments to an hour the cough ceases, free perspiration follows, and the child falls asleep.

Two or three similar attacks may occur in the same night, but on the following day the child may appear quite well. A recurrence of the seizures for several successive nights is not infrequent.

Diagnosis.—*Membranous or Pseudomembranous Croup* (*Diphtheria*).—Hoarseness and dyspnea develop gradually, and the latter is not intermittent. False membrane may be seen in the throat or may be coughed up. The constitutional symptoms are more severe.

Laryngismus Stridulus.—This is a pure neurosis, and is often associated with rickets. The paroxysms resemble those of false croup, but are associated with a peculiar crowing inspiration, and lack catarrhal symptoms, such as hoarseness and cough.

Prognosis.—The prognosis is good.

Treatment.—A sponge moistened with hot water may be applied to the throat, or the child may be placed in a hot bath. If these simple measures fail, an emetic will almost invariably bring relief. Wine of ipecac (1 dram) may be selected. Subsequent treatment should be directed to the laryngeal catarrh. A mixture like the following will be found useful:

℞.	Tincturæ acóniti	♏xx
	Vini ipecacuanhæ	fʒj
	Potassii bromidi	ʒss
	Potassii citratis	ʒj
	Syrupi tolutani	f℥j
	Aquæ	q. s. ad f℥ij.—M.

SIG.—A teaspoonful every two or three hours for a child of two years.

MEMBRANOUS CROUP

(Croupous Laryngitis; True Croup; Pseudomembranous Laryngitis)

See Laryngeal Diphtheria.

CHRONIC LARYNGITIS

Simple Chronic Catarrhal Laryngitis.—This may follow an acute attack, or it may develop gradually from overuse of the voice, excessive smoking, or inhalation of dust or irritant vapors.

Symptoms.—These consist in moderate hoarseness, aphonia after continued speaking, slight cough, and scanty expectoration of grayish mucus tinged with dust or other impurities.

Laryngoscopic examination reveals redness and swelling of the vocal cords or of the entire larynx.

Treatment.—The use of the voice should be restricted. Coexisting nasal or pharyngeal disease should receive attention. The patient must learn to use the voice properly, expelling sounds by the abdominal muscles and diaphragm, and not by the muscles of the throat. Flannel protectors should be avoided, and the application of cool water to the neck, night and morning, instituted in their stead. Tonics are frequently indicated.

Thorough cleansing of the nose, throat, and larynx should be secured by means of mild alkaline sprays (Dobell's solution). Astringent sprays like the following are useful; Zinc acetate, 3 to 5 grains to the ounce; zinc sulphocarbolate, 2 to 3 grains to the ounce; alum, 3 to 5 grains to the ounce. Direct applications of silver nitrate (3 to 5 grains to the ounce) are also very efficacious.

Tuberculous Laryngitis.—This may be primary, but it is more often secondary to tuberculosis elsewhere, especially in the lung.

Symptoms.—These consist in hoarseness, aphonia, hacking cough, and pain in the throat, increased by coughing, speaking, and swallowing.

Laryngoscopic Examination.—The mucous membrane is swollen, *pale*, and edematous. The arytenoid cartilages are especially involved, and the membrane between them is often the seat of a hill-like infiltration. Tuberculous ulcers are usually shallow and have a broad base, an irregular outline, and an uneven surface. They are extremely painful.

Treatment.—The general treatment should be that of pulmonary tuberculosis. The parts should be frequently cleansed with alkaline detergent sprays. Terebene, compound tincture of benzoin, or eucalyptol may be used in a respirator or inhaled from the surface of boiling water. Radical treatment consists in rubbing in, under cocain anesthesia, lactic acid (30 to 75 per cent. solutions). Palliative treatment consists in applying cocain in solution (4 to 10 per cent.) or orthoform, anesthesin, or iodoform in powder. The following insufflation is useful:

℞.	Orthoform	gr. j.
	Acidi borici	gr. j
	Morphinæ sulphatis	gr. ⅛.

Syphilitic laryngitis may manifest itself as a catarrhal inflammation, mucous patches, gummatous infiltration, or ulceration. The ulcers are more or less circular, deep, and sharply circumscribed. They are frequently found on the epiglottis. Rapid necrosis and exfoliation of the cartilage may follow. Pain is often slight.

Diagnosis.—The history, the presence of other syphilitic lesions, the deep, clean-cut, rapidly spreading ulcers, the effect of treatment, and the absence of marked pain and of pulmonary lesions well serve to distinguish syphilis from *tuberculosis.*

Treatment.—Constitutional treatment with salvarsan, iodids and mercurials is of the first importance. Local cleanliness should be secured by thorough spraying with some alkaline antiseptic solution. Ulcers may be touched with silver nitrate (melted on a silver probe), acid nitrate of mercury (1 to 5 parts of water), or chromic acid (1 to 8 parts of water). Insufflations of iodoform are also useful. Cicatricial stenosis may call for gradual dilatation or even tracheotomy.

LARYNGISMUS STRIDULUS

(Spasm of the Glottis; Laryngospasmus; "Child-crowing")

Definition.—A paroxysmal neurosis, characterized by reflex spasm of the adductors of the larynx, and not excited by any local inflammation.

Etiology.—It usually occurs in rachitic infants between six months and two years of age, and is apparently related to tetany. The attacks may be brought on by fright, gastric irritation, exposure, or a sudden movement.

Symptoms.—The attacks often occur on waking from sleep, and are characterized by a sudden arrest of breathing and tonic muscular spasms. The face is pale, and later cyanosed; the eyes are rolled up; the body is arched; the thumbs are turned into the palms; the legs are extended, and the soles turned inward. In a few seconds the spasm relaxes, and air is drawn through the glottis with a shrill, crowing sound.

The seizures vary greatly in frequency: several may occur in a day, or they may be weeks apart.

Diagnosis.—The intermittent character of the affection; the peculiar crowing inspiration; the absence of fever, cough, and hoarseness will serve to distinguish laryngismus from *spasmodic croup.*

Prognosis.—The prognosis is, as a rule, good. In the very young death may result from suffocation.

Treatment.—*The Paroxysm.*—Cold water may be dashed on the face and head, or a few drops of nitrite of amyl may be placed on a handkerchief and held before the nose.

The Interval.—Careful search should be made for some exciting cause; the gums may require lancing, or the gastrointestinal tract may demand attention. The child should be placed under the best hygienic conditions. The food should be plain and nutritious; tonics, like cod-liver oil, hypophosphites, and arsenic, are generally indicated. The bromid of sodium is an efficient antispasmodic, and may be advantageously combined with antipyrin:

℞.	Sodii bromidi	ʒiss
	Antipyrinæ	gr. xx–xxx
	Glycerini	f℥ss
	Aquæ menthæ piperitæ	f℥iij.

Sig.—A teaspoonful four times a day.

EDEMA OF THE LARYNX

(Edema of the Glottis)

Definition.—An infiltration of serous fluid into the submucous tissue of the larynx.

Etiology.—It occasionally results from severe attacks of catarrhal laryngitis. It may be induced by severe inflammation of adjacent organs—as the tonsils, parotid glands, and pharynx. It may be a complication of some acute infectious disease—like diphtheria, scarlet fever, or facial erysipelas. It is sometimes associated with ulcerative affections of the larynx, like tuberculosis and syphilis. It may be excited by the irritation of burns, scalds, or caustics. It occasionally occurs abruptly in the course of Bright's disease.

Pathology.—The connective tissue of the larynx is infiltrated with a serous or seropurulent fluid. The mucous membrane is tense and changed in color.

Symptoms.—These are: hoarseness of the voice, and later aphonia; extreme dyspnea, at first on inspiration, but later on expiration also; stridulous respiration; barking cough; and the evidences of dyspnea—namely, anxious face, blue lips, prominent sternocleidomastoids, and retraction of the base of the chest. When the epiglottis is involved, the swelling can be detected by the finger in the throat.

Laryngoscopic Examination.—The mucous membrane is swollen and of a reddish-purple color. The epiglottis may resemble a round, translucent tumor. In infraglottic edema the upper part of the larynx may appear normal, but swollen and edematous membrane is seen projecting through the glottis. The vocal cords are rarely affected.

Prognosis.—The condition is often fatal.

Treatment.—Mild inflammatory edema sometimes yields to the sucking of ice, local bloodletting, the application of ice to the neck, astringent sprays (alum, adrenalin, tannic acid), and the administration of saline purges. When the symptoms become urgent, the parts should be scarified under cocain anesthesia, and if this fails, tracheotomy should be performed at once.

DISEASES OF THE BRONCHI AND LUNGS

BRONCHITIS

Definition.—An inflammation of the bronchial tubes, characterized by substernal soreness, cough, mucopurulent expectoration, and dry and moist râles.

Varieties.—(1) Acute catarrhal bronchitis; (2) chronic catarrhal bronchitis; (3) fibrinous bronchitis.

ACUTE CATARRHAL BRONCHITIS

Etiology.—A cold, damp climate; changeable weather; occupations that necessitate confinement or the inhalation of irritating dusts or vapors; the gouty diathesis; and chronic heart disease are general predisposing factors.

In many cases the disease follows exposure to cold and wet, particularly when the body is overheated, or the inhalation of irritating gases or dusts. Not rarely it is one of the manifestations of a general infection such as measles, whooping-cough, typhoid fever, influenza, etc.

The exciting cause may be the micrococcus catarrhalis, pneumococcus, influenza bacillus, streptococcus or staphylococcus.

Pathology.—In most cases the trachea and large tubes only are affected. The mucous membrane is red, swollen, injected, and covered in places with tenacious mucopus.

Microscopic examination reveals desquamation of epithelium and infiltration of the submucous tissues with leukocytes.

Symptoms.—The chief features are: Chilliness and general malaise; a sense of soreness and constriction behind the sternum, increased by coughing; slight fever (100° to 102° F.), with its associated symptoms; and cough, which is at first dry and painful, but later accompanied by more or less abundant mucopurulent expectoration.

Physical Signs.—If the inflammation is confined to the trachea and main bronchi there may be no abnormal signs. In other cases auscultation may reveal sonorous and sibilant râles on both sides of the chest in the early stages, and moist râles later when the bronchial secretion is more abundant.

Diagnosis.—*Influenza.*—High fever, severe pain in the head, back, and limbs, and great prostration will serve to distinguish influenza from bronchitis when the former is prevalent.

Catarrhal Pneumonia.—Moderately high and irregular fever, prostration, pronounced dyspnea, cyanosis, and physical signs indicating consolidation will aid in the recognition of pneumonia.

Prognosis.—The prognosis in general is good. In the aged the very young and the debilitated, however, the disease may lead to capillary bronchitis (catarrhal pneumonia).

Treatment.—If the patient be weak or old, he should be confined to his room or even to bed. At the outset, free diaphoresis is useful. It may be secured by means of hot drinks, a full dose of Dover's powder, and a hot foot-bath. Counterirritation to the chest in the form of sinapisms or stupes is very beneficial. The food should be simple and readily digestible, and the bowels should be kept regularly open by the aid of mild aperients. In the early stages, when there is no secretion, sedative expectorants—ipecacuanha, potassium citrate, and tartar emetic—are indicated. It is usually necessary to add a sedative, like opium or one of its derivatives (codein, $\frac{1}{8}$ to $\frac{1}{6}$ grain, or heroin, $\frac{1}{16}$ to $\frac{1}{10}$ grain) to allay the distressing cough. A combination like the following will be found useful:

℞.	Potassii citratis	ʒiij
	Vini ipecacuanhæ	fʒiiss
	Tincturæ opii camphoratæ	fʒiij
	Succi limonis	f℥j
	Syrupi	q. s. f℥vj.—M.

SIG.—A tablespoonful every four hours.

When the secretion becomes more abundant, stimulant expectorants are indicated. One of the most reliable members of this class is ammonium chlorid; it may be prescribed in some simple vehicle, like brown mixture, or with squills, as in the following formula:

℞.	Ammonii chloridi	ʒiiss
	Syrupi scillæ	f℥v
	Tincturæ opii deodorati	♏xl
	Extracti glycyrrhizæ	ʒj
	Glycerini	f℥ss
	Aquæ	q. s. ad f℥iv.—M.

Sɪɢ.—A dessertspoonful in water every four hours.

Among other useful stimulant expectorants may be mentioned terpin hydrate, terebene, oil of eucalyptus, oil of santal, and tar. Such combinations as the following are often serviceable when the catarrh tends to become subacute:

℞.	Terebeni	
	Olei santali	āā f℥ss
	Strychninæ sulphatis	gr. ¼
	Codein. sulphatis	gr. 1½–ij.—M.

Pone in capsulas No. xij.

Sɪɢ.—One every four hours.

Or:

℞.	Ammonii chloridi	ʒj
	Vini picis liquidæ	
	Misturæ glycyrrhizæ compositæ	āā f℥iss.—M.

Sɪɢ.—A dessertspoonful every two or three hours. (Mᴜssᴇʀ.)

In the aged and infirm alcoholic stimulants may be required to combat general adynamia. Strychnin is a most valuable adjunct to the expectorants when there are indications that the heart is becoming strained by the violent paroxysms of cough. Should there be evidence of pronounced cardiac failure, it will be necessary to employ digitalis.

Such tonics as cod-liver oil, iodid of iron, quinin, and arsenic are often useful during convalescence from severe and prolonged attacks. Much benefit will also be obtained from suitable change of climate.

CHRONIC BRONCHITIS

(Chronic Bronchial Catarrh; Winter Cough)

Etiology.—This condition may be the result of repeated attacks of acute bronchitis, or it may develop gradually in association with chronic cardiac, pulmonary, or renal disease, or gout. It is especially common in the aged.

Pathology.—The mucous membrane of the bronchi is sometimes thickened and granular; in other cases it is thin from atrophic changes. The surface is usually covered with mucopus; ulcers are occasionally noted.

Long-standing bronchitis leads to dilatation of the tubes (bronchiectasis) and to emphysema.

Symptoms.—The chief features are: Persistent cough with more or less mucopurulent expectoration; a sense of soreness behind the sternum. Fever is usually absent, and unless the disease is very severe, the general health may be fairly well preserved. Dyspnea on exertion is sometimes a troublesome symptom; it, however, belongs more to the resulting emphysema than to the bronchitis.

PHYSICAL SIGNS.—Unless emphysema has developed, inspection, palpation, and percussion give negative results.

Auscultation sometimes reveals râles, some of which are dry and wheezing, while others are moist and bubbling.

Bronchorrhea.—This term is applied to cases of chronic bronchitis which are associated with a very copious expectoration. The sputum is generally mucopurulent, and sometimes very offensive (fetid bronchitis.)

Dry Catarrh.—This form, described by Laennec as *catarrhe sec*, is characterized by severe paroxysms of coughing with little or no expectoration. It is generally seen in the aged in association with emphysema or asthma.

Diagnosis.—*Pulmonary Tuberculosis.*—The absence of fever, of hemorrhage, of bacilli in the sputa, and of signs indicating consolidation will serve to distinguish chronic bronchitis from pulmonary tuberculosis.

Bronchiectasis.—This often results from chronic bronchitis. It is characterized by paroxysms of cough attended with the expectoration of large quantities of purulent secretion of an extremely offensive odor. There may be, also, physical signs of one or more cavities near the root or base of the lung.

Emphysema.—Marked dyspnea, distention of the chest, hyperresonance on percussion, and a prolonged feeble expiration on auscultation will indicate emphysema.

Sequelæ.—These comprise emphysema, bronchiectasis and dilatation of the heart, especially of the right ventricle.

Prognosis.—Complete recovery is, as a rule, not attainable, but the disease is not incompatible with long life.

Treatment.—Treatment must be directed toward the prevention of recurring acute attacks, and the removal, if possible, of the underlying cause. Change of climate, especially in winter, is most beneficial. If there is much secretion, a dry, warm climate is generally to be recommended, whereas if there be little expectoration, a moist warm climate is preferable. If patients cannot afford to travel, they should remain indoors as much as possible in bad weather, and take every precaution against chilling. Flannel should at all times be worn next to the skin, the feet should be kept dry, and night air should be avoided.

Underlying chronic diseases should receive appropriate treatment. If cardiac insufficiency is present, digitalis or strychnin will be required. If there is general malnutrition, such remedies as iron, arsenic, cod-liver oil, and hypophosphites may be given with advantage. If gout is a factor, iodids and alkalis will prove serviceable.

The most useful *direct remedies* are the stimulant expectorants, such as terebene, oil of eucalyptus, myrtol, oil of santal, oil of copaiba, oil of cubeb, and tar. If the sputum is heavy and purulent, no drug acts so well as creosote or the carbonate of guaiacol. Potassium iodid may be tried if the expectoration is scanty and viscid. Mild anodynes—heroin or codein—

are often necessary to control harassing cough. The following formulas will illustrate the manner in which these remedies may be combined:

℞. Terebeni
Olei eucalypti
Olei santali . . . . āā fʒj-iss
Codeinæ sulphatis . . . . gr. iij-vj.—M.
Pone in capsulas No. xxiv.
Sig.—One after each meal and at bedtime.

℞. Terpini hydratis . . . . ʒj
Guaiacolis carbonatis . . . . ʒij
Strychninæ sulphatis . . . . gr. ss
Codeinæ sulphatis . . . . gr. iij.—M.
Pone in capsulas No. xxiv.
Sig.—One or two capsules three or four times a day.

℞. Apomorphinæ hydrochloridi . . . . . gr. ss
Syrupi pruni virginianæ . . . . f℥ij
Syrupi picis liquidæ . . . . f℥iv.—M.
Sig.—A tablespoonful thrice daily. (Murrell.)

Inhalations (eucalyptol, terebene, oil of Scotch fir, compound tincture of benzoin, etc.) are often efficacious. Such a mixture as the following may be employed several times a day in an oronasal respirator:

℞. Chloroformi . . . . fʒss
Creosoti
Terebeni
Olei pini sylvestris . . . . āā fʒiss
Alcoholis . . . . q. s. ad f℥j.
Sig.—From 5 to 20 drops to be used in the inhaler several times a day.

Counterirritation, preferably with iodin or small blisters, is often of great service in lessening the severity of acute exacerbations.

FIBRINOUS BRONCHITIS

Definition.—A rare affection characterized by the expectoration of fibrinous casts of certain portions of the bronchial tree.

Etiology.—The causes are unknown. In some cases it has been associated with tuberculosis, while in others there has been chronic heart disease.

Pathology.—The disease is often limited to a certain number of bronchi. Some of the affected tubes are found filled with a fibrinous exudate, while others are found empty and show a loss of epithelium. The casts are usually expelled in the form of whitish balls, which, when unrolled in water, present branching molds of the divisions and subdivisions of the affected bronchi. On close examination they are found to be hollow and laminated. Under the microscope, a homogeneous or fibrillated membrane is observed, imbedded in which are leukocytes, fat-drops, particles of pigment, epithelial cells, and occasionally Leyden's octahedral crystals.

Symptoms.—Acute and chronic forms are recognized. The former is rare, and manifests the symptoms of a severe attack of acute bronchitis, but the sputa contain fibrinous casts and there is marked dyspnea.

The chronic form is characterized by severe cough, paroxysms of dyspnea, and the expectoration of fibrinous plugs. Hemoptysis is not uncommon. The physical signs are those of chronic bronchitis and emphysema. The disease often lasts a few weeks, and then disappears, to return again at definite periods.

Prognosis.—In the acute variety the prognosis must be guarded; death frequently results from suffocation. In the chronic form attacks occur at intervals through many years.

Treatment.—In the acute cases the atmosphere of the room should be kept moist and uniformly warm. Inhalations of alkaline vapors (limewater) appear to be beneficial. Counterirritants should be applied to the chest. Emetics sometimes aid in the expulsion of loose casts. Sedative or stimulant expectorants may be prescribed, as in catarrhal bronchitis. In the chronic form potassium iodid may also be given.

BRONCHIECTASIS

(Dilatation of the Bronchi)

Etiology.—Bronchiectasis is most frequently the result of chronic bronchitis, weakening of the walls of the bronchi from the inflammation and increased pressure from the violent coughing being the determining factors. In children it is sometimes secondary to bronchopneumonia. In other cases it develops as a sequel to chronic interstitial pneumonia, tuberculosis, or chronic dry pleurisy. Occasionally the disease is caused by obstruction to a bronchus (foreign body, aneurysm, tumor, etc.), and in some cases it appears as a congenital defect.

Pathology.—Two forms are noted: (1) The cylindric form, in which the tubes, particularly those of medium size, are uniformly dilated in one or both lungs; and (2) the saccular form, in which the tubes swell out, here and there, into circumscribed dilatations that may reach several inches in diameter. Bronchiectatic cavities are lined with mucous membrane, but the latter is often atrophied, indurated, or ulcerated.

Symptoms.—The chief symptoms are paroxysmal cough, dyspnea, and copious expectoration. The last is characteristic. From time to time, especially in the morning or on a sudden change of posture, the patient expels a large amount (200–600 c.c.) of extremely fetid mucopurulent sputum, which on standing separates into three layers: an upper layer of discolored froth, a middle layer of turbid mucus, and an under layer of decomposed pus. Microscopically, it contains pus-corpuscles, fat crystals, crystals of hematoidin, and numerous micro-organisms, but no tubercle bacilli. Elastic fibers are rarely found. Hemoptysis is not uncommon.

PHYSICAL SIGNS.—In the cylindric variety the signs are those of chronic bronchitis. The saccular variety may present the signs of tuberculous cavities—localized tympany, cavernous breathing, gurgling râles, and pectoriloquy. Bronchiectatic cavities are usually near the root or the base of the lung. In long-standing cases the fingers are often clubbed.

Diagnosis.—*Pulmonary Tuberculosis.*—The marked constitutional symptoms, the apical location of the cavities, the signs of consolidation around the cavities, and the presence of tubercle bacilli in the sputum will establish the diagnosis.

Prognosis.—There is little prospect of cure, but life may be prolonged indefinitely. Abscess or gangrene of the lung, metastatic abscesses, especially in the brain, and amyloid disease of the viscera are not uncommon sequels.

Treatment.—The general treatment is that of chronic bronchitis. The most useful expectorants are oil of eucalyptus, oil of santal, terebene, tar, guaiacol carbonate, and creosote. Inhalations of terebene, carbolic acid, creosote, etc., lessen cough and aid in destroying the fetid odor of the breath. Intratracheal injections (2–3 drams of olive oil containing 1 or 2 per cent. of guaiacol) are sometimes efficacious. If a single large cavity can be definitely located in the lower lobe, incision and drainage may be considered.

ASTHMA

Definition.—Paroxysmal dyspnea due to spasm of the bronchi or to sudden swelling (turgescence) of the bronchial mucosa.

Dyspnea of this type may occur symptomatically in chronic nephritis (*renal asthma*) or in chronic heart disease (*cardiac asthma*); or it may occur at intervals for years or throughout life, independently of any other affection. As a primary condition it may be due to the action of the pollen of certain grasses or plants upon a hypersensitive respiratory mucosa, and when induced in this way it is known as *hay asthma* or *hay fever* (see p. 274); or it may be brought on by the action of divers irritants, the nature of which is often obscure, and when this is the case it is known as *bronchial asthma.*

BRONCHIAL ASTHMA

Etiology and Pathogenesis.—The disposition to asthma is sometimes hereditary. No age is exempt, but the majority of cases begin in early life and few begin after fifty. Males are more frequently affected than females. In many cases there is associated disease of the nasal mucosa or accessory nasal sinuses—polyps, hypertrophic rhinitis, adenoids, antral suppuration, etc. Some cases appear to be related etiologically to functional disturbances of the stomach or intestines.

In asthmatic subjects the paroxysms may be excited by irritating dust, emanations from certain animals (particularly the horse) or plants, certain articles of food, overeating, exposure to cold and damp, violent emotion, etc.

The chief pathologic conditions exhibited in an attack appear to be spasm of the bronchial muscles and congestive swelling (turgescence) of the mucous membrane of the bronchioles, and the theory is gaining in favor that these changes are anaphylactic manifestations, due to the action of some foreign protein, sensitization of the subject having taken place by previous entrance of the particular protein into the blood or by heredity.

Symptoms.—The paroxysms often appear suddenly, but in some cases certain symptoms precede and give warning of the approaching attack; among these are chilliness, flatulence, sneezing, and a copious discharge of pale urine. The attacks most often occur at night. There is a sense of oppression and anxiety, followed by dyspnea so intense that the patient runs to the window for air, or sits upright with his arms in such a position that he can bring into play the auxiliary muscles of respiration. The face is pale and anxious, the lips are blue, and the surface is covered with profuse perspiration. The respirations are not rapid, but labored and noisy. Cough is usually present, and is associated with the expectoration of thick, tenacious mucus. On close examination little grayish plugs can be detected in the sputum. These, under a pocket-lens, are seen to consist of delicate spirals of mucus that have been molded in the finer bronchioles (Curschmann's spirals). In addition, the sputum contains many eosinophilic leukocytes and Charcot-Leyden octahedral crystals. Examination of the blood usually reveals eosinophilia.

The paroxysms last, as a rule, two or three hours, and may recur for several successive nights, or may disappear entirely for weeks or months.

Physical signs are definite. During the attack the chest is fixed in full inspiratory expansion, and the abdomen is immobile. *Percussion* gives hyperresonance in long-standing emphysematous cases. On *auscultation* there are numerous sonorous and sibilant râles, especially during expiration, which is greatly prolonged and wheezy. Toward the end of an attack the râles become moist.

Diagnosis.—*Cardiac and renal asthma* are to be distinguished from bronchial asthma by the history and by the evidence of organic heart or kidney disease.

Hay asthma may be recognized by the periodicity of the attacks and the associated coryza and sneezing.

Laryngeal Obstruction from foreign bodies, croup, paralysis of the vocal cords, or edema.—The dyspnea is with inspiration, and the chest, instead of being distended, is retracted, especially at the base.

Sequelæ.—Emphysema invariably follows if the asthma is of long duration; it results from the tension to which the vesicles are subjected during the expiratory effort. Dilatation of the right ventricle is also a remote sequel.

Prognosis.—The disease does not prove fatal except through complications or sequelæ. Complete recovery may occur in young subjects if a local cause can be found and removed.

Treatment.—*The Interval.*—The cause must be sought for in every case, and removed if possible. Digestive disturbances should always receive careful attention. Chronic bronchitis, emphysema, and dilatation of the heart are frequent concomitants of asthma and call for special treatment. Change of climate, even though slight, usually proves of decided service, but the choice of locality must be determined very largely by the personal experience of the patient. Many sufferers do better in the smoky atmosphere of cities than in the country, but a dry atmosphere with a moderate elevation is better suited to the majority.

Among internal remedies none has proved more useful than potassium iodid (5 to 10 grains thrice daily) in averting attacks. Tincture of belladonna (3 to 5 minims thrice daily) is a valuable adjunct to the iodid. Arsenic may be tried if the iodids fail. Grindelia is sometimes useful if there is much catarrh. Strychnin is of service in cases associated with emphysema.

The Attack.—Some patients derive great benefit from the fumes of ignited stramonium or belladonna leaves or paper that has been impregnated with potassium nitrate. These agents may be burnt in the patient's room or smoked in a pipe or in the form of cigarettes. Marked alleviation of the paroxysms is often obtained from the inhalation of amyl nitrite (5 to 6 minims), ethyl iodid (10 to 20 minims), or a few whiffs of chloroform. If such measures fail to afford relief, internal remedies must be used. In some cases strong hot coffee acts most happily; in others more benefit is derived from hot whisky and water. Among the numerous special remedies that have been advocated the following appear to be the most reliable: opium, belladonna, bromids, chloral, paraldehyd, Hoffmann's anodyne, lobelia, and quebracho.

Few attacks will resist the action of morphin with atropin hypodermically, but the greatest caution must be exercised in order that the patient may not become addicted to the drug. Heroin hydrochlorid hypodermically, in doses of from 1/12 to 1/10 grain, may often be substituted for morphin with advantage. In some cases hypodermic injections of adrenalin (15 minims) are very serviceable. When the attacks are associated with bronchial catarrh, a combination like the following sometimes proves efficacious:

℞.	Tincturæ belladonnæ	fʒj
	Tincturæ lobeliæ	fʒiij
	Fluidextracti aspidospermatis	f℥ss
	Spiritus ætheris compositi	fʒv
	Strontii bromidi	ʒiiss
	Elixiris aromatici	q. s. ad f℥iv.—M.

SIG.—A dessertspoonful in water every two or three hours.

Among other measures that have been found useful in alleviating asthmatic attacks may be mentioned the application of sinapisms to the chest, the inhalation of compressed air, and the inhalation of oxygen.

HAY ASTHMA

(Hay Fever; Autumnal Catarrh; Rose-cold)

Definition.—A catarrhal affection of the upper air-passages, characterized by coryza and asthmatic seizures, and evoked by inhaling the pollen of certain plants and grasses.

Etiology and Pathogenesis.—An inherited tendency, a nervous temperament, and chronic nasal disease are predisposing factors. The attacks, as a rule, occur in the autumn (autumnal catarrh) or in the spring (rose-cold), and are excited by certain plants and grasses (ragweed, goldenrod, timothy grass, etc.).

The disease appears to be a local manifestation of hypersensitiveness (anaphylaxis) to the toxalbumin of the pollen (see p. 271).

Symptoms.—Nasal obstruction with rhinorrhea and much sneezing, congestion of the conjunctivæ with lacrimation, itching of the eyelids, nose, and palate, headache and lassitude, and, not rarely, paroxysms of asthma, constitute the symptoms.

"Rose-cold" usually begins in May or June and lasts to the latter part of July; "autumnal catarrh" begins in the latter part of August and ends with the first frost.

Prognosis.—The disease never proves fatal, but, as a rule, it persists throughout life.

Treatment.—Careful search should be made for chronic nasal disease, and, if found, appropriate treatment instituted.

A change of climate during the period of susceptibility exempts most patients. A sea-voyage or a sojourn in some high-mountain district, like the White Mountains, Adirondacks, Catskills, or Alleghanies, may be recommended.

Tonics such as quinin, arsenic, and strychnin are often very useful when administered before and during an attack. To allay itching and lacrimation the eyes may be washed with a solution of boric acid (10 grains to the ounce) or sulphate of zinc (1 to 2 grains to the ounce). Sneezing, nasal fulness, and discharge are often relieved by medicated sprays

(Dobell's solution) or the application, on pledgets of cotton, of adrenalin solution (1:5000). In some cases good results have been obtained from subcutaneous injections of very dilute extracts of the special pollen. Treatment by antitoxic serum produced by the method of Dunbar is of doubtful value.

PULMONARY EMPHYSEMA

Definition.—Abnormal distention of the lungs with air.

Varieties.—(1) *Interlobular Emphysema.*—This form is rare, and results from the rupture of the air-vesicles and the escape of air into the interstitial tissue.

(2) *Compensatory Emphysema.*—This is a vicarious distention of one part of the lung, owing to pathologic changes in another part of the organ. It is primarily physiologic, though atrophy of the walls of the air-vesicles may ultimately ensue.

(3) *Atrophic or Senile Emphysema.*—In this form the capacity of the air-vesicles is relatively increased, owing to atrophy of the solid tissue.

(4) *Hypertrophic or Substantive Emphysema.*—This is the ordinary form of emphysema. It is characterized by a great enlargement of the lungs in consequence of overdistention of the air-vesicles.

Compensatory emphysema, atrophic emphysema, and hypertrophic emphysema together form a subdivision known as vesicular emphysema.

HYPERTROPHIC EMPHYSEMA

Definition.—A pulmonary disease characterized anatomically by dilatation of the air-vesicles and atrophy of their walls; and clinically by permanent enlargement of the thorax, with more or less dyspnea.

Etiology.—Congenital weakness of the lung structure—probably a defective development of elastic tissue—is an important predisposing factor. This predisposition may be transmitted through several generations.

In forced expiration the air cannot escape with sufficient rapidity through the narrow glottis, and the backward pressure stretches the air-vesicles; hence the obstinate cough of chronic bronchitis, the expiratory straining of asthma, and occupations that necessitate forced expiration, like playing on wind-instruments and glass-blowing, are causal factors.

Pathology.—The lungs are enlarged and do not collapse when the thorax is opened. In the advanced cases the free margins are studded with large bullæ or blebs that have resulted from the rupture of a number of vesicles into a common sac. The organs are pale and have a soft, cotton-like feel. Microscopic examination reveals atrophy of the vesicular walls, a diminished amount of elastic tissue, and more or less obliteration of the pulmonary capillaries. This last condition leads to increased tension in the pulmonary artery and ultimately to enlargement of the right ventricle.

Symptoms.—The disease usually manifests itself in middle life, but it is sometimes observed in the young. Dyspnea, at first only upon exertion, but as age advances, more or less persistent; a disposition to bronchitic attacks with cough and expectoration upon the slightest exposure; and cyanosis, often extreme during attacks of acute bronchitis are the usual symptoms. In advanced cases edema of the feet may result from cardiac failure.

PHYSICAL SIGNS.—The neck is short, and the sternocleidomastoids are prominent. The thorax is likewise short but broad, especially in its anteroposterior diameter. This configuration has given rise to the term "barrel-shaped" chest. On respiration there is little expansion, but an elevation of the thorax as a whole. The apex-beat is usually invisible, but an abnormal pulsation is often noted in the epigastrium.

Palpation.—Vocal fremitus is usually diminished.

Percussion.—This yields hyperresonance all over the chest. The upper level of hepatic dulness is depressed, and the area of cardiac dulness may be almost obliterated.

Auscultation.—In uncomplicated emphysema the breath sounds are feeble and the expiration is much prolonged. In the presence of bronchitis, however, the breath sounds may be harsh or marked by coarse râles. The pulmonary second sound is often accentuated.

Complications.—The most important complications are: Bronchitis, asthma, dilatation of the right ventricle, and, later, tricuspid regurgitation and dropsy.

Diagnosis.—*Chronic Bronchitis.*—In this disease there is no marked dyspnea; the chest is not enlarged; there is no change in the percussion-note or in the expiratory sound.

Pneumothorax.—This disease develops suddenly, is unilateral, and yields a tympanitic note on percussion and, frequently, metallic tinkling and bell-tympany on auscultation.

Prognosis.—Emphysema is incurable, but its advance may be stayed by relieving the primary condition. It runs a long course and is in itself rarely fatal, but death may result from heart-failure and dropsy, or from intercurrent pneumonia.

Treatment.—The treatment advocated in chronic bronchitis and asthma is often applicable here. The patient should be placed under the most favorable hygienic conditions. Iron is indicated in the anemic. Strychnin (1/40 to 1/30 grain) is a valuable respiratory and cardiac stimulant, and may be combined with digitalis when there are symptoms of heart-failure.

℞. Strychninæ sulphatis . . . . gr. ss
Pulveris digitalis
Pulveris scillæ
Ferri reducti. . . . . āā gr. xx.—M.
Fiant pilulæ No. xx.
SIG.—One thrice daily.

The inhalation of oxygen, or the inspiration of compressed air, followed by expiration into rarefied air, is sometimes a useful measure.

HEMOPTYSIS

(Bronchorrhagia; Bronchopulmonary Hemorrhage)

Definition.—The expectoration of blood.

Etiology.—The chief causes are: (1) Traumatism. (2) Certain organic diseases of the lung, especially tuberculosis (80 per cent. of the cases), lobar pneumonia, bronchiectasis, gangrene, infarct, and cancer. (3) Passive congestion the result of heart disease, especially mitral lesions. (4) Aortic aneurysm, from erosion of the trachea or a bronchus, or rupture of the sac into the respiratory tract. (5) Diseases profoundly affecting the blood, such as purpura, hemophilia, scurvy, and leukemia. (6) Ulcers, traumatic, syphilitic, or malignant, of the trachea or larynx. (7) Vicarious menstruation (very rare).

Symptoms.—Sometimes the bleeding is preceded by cough, dyspnea, or substernal tenderness, but often there is no premonition, and the first indication is the presence of a warm salty fluid in the mouth. The blood is generally raised by coughing, and is bright red and frothy. It is alkaline in reaction, and intimately mixed with air and mucus. The hemorrhage is rarely profuse unless it results from the rupture of an aortic aneurysm or the ulceration of a large vessel in advanced pulmonary tuberculosis. Auscultation of the chest reveals bubbling râles. The subsequent expectorations are tinged with blood, and if much is swallowed, it may excite vomiting or pass into the intestine and impart a tarry appearance to the stools.

Diagnosis.—The differential diagnosis between *hematemesis* and hemoptysis has been considered on page 67.

Prognosis.—This depends upon the cause. It is rarely fatal, except in aneurysm and in advanced tuberculosis with a large cavity.

Treatment.—Absolute rest is essential. An ice-bag may be placed over the suspected seat of the hemorrhage, but it should be removed if it aggravates the coughing. Bits of ice may be given to the patient to suck. There is no more useful remedy than morphin, which serves to ally excitement and to check cough. It is best given hypodermically. Nitroglycerin (1 drop every half hour) has been strongly recommended. If the hemorrhage is protracted, a saline purge may be useful. Among other remedies that seem to be of service may be mentioned oil of erigeron, fluid extract of hamamelis, and gelatin. Ergot is useless, and so is the inhalation of vaporized solutions of astringent drugs. The application of firm ligatures to the limbs may prove efficacious by lowering the intrapulmonary pressure.

HEMORRHAGIC INFARCT OF THE LUNG

(Pulmonary Apoplexy)

Definition.—A circumscribed area of necrosis infiltrated with blood.

Etiology.—The most common cause of pulmonary infarct is obstruction of a branch of the pulmonary artery by an embolus coming from the right heart or the general venous system. In some cases the obstruction is caused by a thrombus, the formation of which has been favored by cardiac weakness.

Pathology.—The infarct is usually located in the periphery of the lung; it is conic in shape, with its apex pointing inward. The portion affected is firm, airless, and of a dark red color. Microscopic examination shows a dense aggregation of blood-corpuscles.

In small infarcts the dead tissue and blood are slowly absorbed and replaced by a cicatrix.

Symptoms.—When the infarct is large, the usual symptoms are localized pain, dyspnea, cough, and the expectoration of dark non-aërated blood. These symptoms occurring in chronic heart-disease or phlebitis are especially suggestive. Small infarcts occasion no *physical signs;* larger ones may yield a circumscribed area of dulness, with subcrepitar long time, the organs become brown, dense, and tough (*brown induration*). Microscopic examination reveals dilatation of the capillaries, overgrowth of connective tissue, brown pigmentation, and degenerative changes in the blood-vessels.

Symptoms.—Dyspnea, cough, and the expectoration of blood-stained mucus containing pigmented epithelial cells (heart-disease cells) are the characteristic symptoms. *Physical examination* reveals slight dulness, feeble breathing, and abundant fine râles.

Treatment.—Remedies should be directed to the underlying cardiac disease. The application of dry or wet cups often gives temporary relief. If the symptoms are urgent, venesection is indicated. Saline and mercurial aperients are of service.

HYPOSTATIC CONGESTION

Definition.—A congestion of dependent portions of the lungs occurring in asthenic diseases that necessitate a protracted recumbent position.

Etiology.—It is generally observed in prolonged febrile diseases, such as typhoid fever, and in adynamic states. Cardiac weakness, recumbent posture, and alterations in the blood are the causal factors.

Pathology.—The lungs are dark red and edematous posteriorly. The edema and increased amount of blood render the organs more solid and less crepitant. They never show the granular appearance of croupous pneumonia.

Symptoms.—The symptoms are often indefinite. There may be moderate dyspnea, slight cyanosis, cough, and, perhaps, blood-tinged expectoration.

Physical examination reveals dulness over the lower lobes, subcrepitant râles, and feeble, blowing breathing.

Treatment.—Efforts should be made to prevent the development of hypostatic pneumonia in asthenic diseases by frequent change of posture and the timely administration of such stimulants as strychnin, digitalis, alcohol, ammonia, and camphor. If already present, relief is sometimes afforded by dry or wet cupping or the application of stupes.

EDEMA OF THE LUNGS

Definition.—An effusion of serous fluid into the air-vesicles and interstitial tissue of the lungs.

Etiology.—Pulmonary edema may occur under the following conditions: (1) In passive congestion of the lungs, the result of chronic heart disease; (2) as a part of general dropsy induced by Bright's disease; (3) as a terminal process in many acute and chronic disorders; (4) in ether anesthesia; (5) after thoracentesis; (6) as an acute disorder in chronic disease of the heart, arteries, or kidneys and in certain infectious diseases; (7) around circumscribed lesions of the lung, such as pneumonia, tuberculosis, infarct, etc. (collateral edema).

Pathology.—The lungs, especially the dependent portions, are heavy, red in color, and boggy to the feel. When the affected portion is incised and subjected to pressure, abundant blood-stained, frothy serum exudes.

Symptoms.—These consist in dyspnea sometimes amounting to orthopnea, cyanosis, cough, and the expectoration of large quantities of frothy, serous fluid. Occasionally the sputum is blood-stained. The skin is often cold and livid. There is no fever.

Physical examination reveals feeble tactile fremitus, dulness, weak breath-sounds, and numerous fine and coarse, moist râles.

Diagnosis.—*Croupous Pneumonia.*—This is characterized by a chill, fever, pain, rusty expectoration, and signs of consolidation.

Hydrothorax.—In this condition there may be enlargement of the affected side, with displacement of the apex-beat. The upper level of dulness is often movable, and frothy sputum and râles are absent.

Prognosis.—This is always grave. It is often a terminal symptom of the disease in which it occurs. If not far advanced, and the primary disease is amenable to treatment, recovery may follow.

Treatment.—If there is much cyanosis and the patient's strength will permit it, the application of wet cups to the chest or bleeding from the arm is of great value. Sinapisms should be applied to the chest. Hydragogue cathartics (Epsom salts) are often indicated. Circulatory stimulants, such as strychnin, caffein, camphor, and digitalis, are generally indicated and may be given hypodermically. Atropin (1/100 grain) with morphin (1/6–1/4 grain), hypodermically, is sometimes of great service. Inhalations of oxygen may be employed to lessen the dyspnea.

CROUPOUS PNEUMONIA

(Lobar Pneumonia; Pneumonitis; Lung Fever)

Definition.—An acute infectious disease with a pulmonary localization, characterized clinically in its ordinary form by high temperature, cough, dyspnea, blood-tinged expectoration, variable toxemia, and physical signs indicating consolidation of one or more lobes of the lung.

Etiology.—The disease occurs at all ages. It is probably most common in early adult life. Males are attacked more frequently than females. The majority of cases occur in the winter and spring months. Lowered vitality from preexisting disease (typhoid fever, influenza, nephritis, arteriosclerosis, diabetes, etc.) or excessive fatigue, exposure to cold, and alcoholism are important predisposing factors. Traumatism has a slight etiologic influence. One attack renders the patient more liable to subsequent infection.

The exciting cause of typical croupous pneumonia in the vast majority of cases is the *Diplococcus lanceolatus* or *pneumococcus* of Fraenkel. Other organisms are rarely present in pure cultures.

Pathology.—Anatomically three stages have been recognized: (1) That of congestion; (2) that of red hepatization; (3) that of gray hepatization.

Stage 1.—The affected portion remains distended when the chest is opened; it is of a deep-red color, and is more resistant to the touch than the normal lung. On section, a frothy, blood-stained serum freely exudes. Microscopic examination reveals a dilated and tortuous condition of the capillaries, swelling of the alveolar cells, and a slight corpuscular exudate.

Stage 2.—The hepatized portion is increased in volume, is quite firm, is of a dark-red color, and so heavy that it sinks in water. It is very friable, and the torn surface is dry and presents a granular appearance, owing to the projection of fibrinous plugs from the alveoli.

Microscopic examination reveals a mesh of coagulated fibrin inclosing numerous red blood-corpuscles and some leukocytes. The latter are also noted in the interlobular tissue. In sections properly treated the diplococcus may be detected.

Stage 3.—The red color gives place to a mottled gray, and the solidified area begins to soften. The change in color is due to the compression of the capillaries, to the disintegration of red corpuscles and their replacement by polymorphonuclear leukocytes, and to fatty degeneration of some of the elements.

The consolidation usually begins at the base and extends upward. The most frequent seat is the lower lobe of the right lung. The bronchi and the adjacent pleura are involved in the inflammatory process.

Events.—Resolution commonly occurs, the exudate first undergoing liquefaction (autolysis) through the agency of a ferment liberated by the leukocytes and then being rapidly removed by absorption. Death may occur at any period of the disease from general toxemia, the severity of which is often altogether disproportionate to the area of lung involved; from dilatation of the right ventricle; from asphyxia; or a pneumococcic complication, such as meningitis or endocarditis.

Abscess, gangrene, and chronic interstitial pneumonia are rare terminations.

Symptoms.—The disease usually begins with a decided *chill* and a sharp *pain* in the side, followed by a rapid *rise of temperature*. The latter often attains its maximum (104°–105° F.) in twenty-four hours, and generally continues high, with slight diurnal remissions, for a period from five to ten days, when it falls by crisis, frequently reaching the normal within twenty-four hours. In protracted cases the temperature not rarely falls by lysis. There is marked dyspnea and the respirations are shallow and rapid, sometimes reaching 60 or even 70 per minute. The pulse is accelerated but not in proportion to the respiration, so that the *pulse-respiration ratio* may be 3 to 1 or 2 to 1, instead of 4 to 1. In unfavorable cases the pulse becomes very frequent (140–160) and weak. *Cough* is a prominent symptom: at first it is short and dry, but later it is accompanied by *bloody or rusty translucent and tenacious sputa.* Microscopically the sputum contains red blood-corpuscles, free pigment, pus-corpuscles, diplococci, and other micro-organisms. The face is flushed; the lips are cyanosed and often the seat of an herpetic eruption; the tongue is heavily furred; the bowels are constipated; the urine is scanty, high-colored, deficient in chlorids, and often slightly albuminous. In severe cases *delirium* is rarely absent. Examination of the blood usually shows marked polynuclear leukocytosis (15,000 to 40,000).

PHYSICAL SIGNS.—*Inspection.*—There may be deficient expansion over the affected side. There is no bulging of the interspaces nor displacement of the apex-beat.

Palpation.—In the vast majority of cases the vocal fremitus is considerably increased over the affected area.

Percussion.—In the earliest stage there may be hyperresonance from diminished intrapulmonary tension. As consolidation advances, however, the note becomes remarkably dull. Percussion over unaffected lobes yields a tympanitic note (Skoda's resonance).

Auscultation.—In the stages of congestion fine crepitant râles are heard at the end of full inspiration. They are probably produced by the forcible separation of adherent vesicular walls. In the stage of consolidation auscultation usually reveals exaggerated vocal resonance and bronchial breathing, but the breath sounds may be absent if the bronchioles are filled with exudation. During resolution the softening of the exudate gives rise to fine moist râles—the *redux crepitus*.

Atypical Cases.—*Senile Pneumonia.*—The symptoms often develop insidiously; the temperature may not be high; the pulse may not be accelerated; expectoration is often absent; the physical signs are not marked; delirium is common; weakness is extreme; and death from exhaustion is the most frequent termination.

Pneumonia in Children.—It is often ushered in with vomiting or convulsions. Headache, delirium, and stupor are prominent symptoms, so that the disease may simulate meningitis. The temperature is very high; expectoration is often absent. The disease frequently begins at the apex of the lung. The duration is usually short.

Typhoid Pneumonia.—In this form there are pronounced typhoid symptoms—headache, muttering delirium, stupor, a dry, brown tongue, subsultus tendinum, carphologia, a rapid, weak pulse, and high fever. The expectoration may resemble prune-juice.

Pneumonia of Drunkards.—The onset is often gradual; the dyspnea is marked; the temperature is not high; maniacal delirium commonly develops; and death from exhaustion is exceedingly frequent.

Massive Pneumonia.—In this form the bronchioles, as well as the air-vesicles, are filled with fibrinous exudate. The physical signs resemble those of pleural effusion.

Central Pneumonia.—In this form the inflammatory process commences in the center of a lobe, and in consequence the characteristic physical signs may not manifest themselves for two or three days.

Migratory Pneumonia.—In this type the specific inflammation shows a tendency to spread and to involve successively fresh areas of lung tissue.

Complications.—These are usually due to pneumococcic infection. Pleurisy is the most common complication. It may be either serous or purulent. Pericarditis and endocarditis are not very infrequent. The latter is sometimes of the malignant type. Among less frequent complications may be mentioned meningitis, arthritis, parotitis, nephritis, catarrhal jaundice, acute dilatation of the stomach, and delayed resolution (consolidation may last for five or six weeks and then gradually disappear). Abscess, gangrene, and chronic interstitial pneumonia are rare sequels.

Diagnosis.—*Pleurisy.*—There is rarely a distinct chill; fever is not so high nor the pulse so rapid; there is no rusty sputum; nervous symptoms are wanting; there is often bulging of the interspaces, with displacement of the apex-beat; the level of dulness may change with the posture of the patient; vocal fremitus and vocal resonance are diminished; and the breath-sounds are usually weak and distant.

Acute Pulmonary Tuberculosis.—The history, the mode of onset, the long duration, the remittent fever, the rapid emaciation, profuse sweats, and presence of tubercle bacilli and elastic fibers in the sputum will suggest tuberculosis.

Pulmonary Edema.—In edema there is absence of chill, fever, and pain; the expectoration is frothy and serous; both lungs are commonly affected; auscultation reveals abundant subcrepitant râles and weak breathing.

Typhoid Fever.—Typhoid pneumonia may readily be mistaken for typhoid fever with pneumonia; but pneumonia as a complication occurs late in the disease, so that the history of the onset gives much assistance. Moreover, in typhoid pneumonia there is no roseolar rash or characteristic blood-serum reaction.

Prognosis.—In young, robust subjects of good habits the prognosis is good. After the age of sixty the outlook is grave. In drunkards the disease is especially fatal. The coexistence of heart or kidney disease makes pneumonia exceedingly dangerous.

In any case a pulse more frequent than 120 per minute, especially if weak and irregular, absence of leukocytosis, pronounced cerebral symptoms, and marked meteorism (toxic paresis of the intestine) are unfavorable features. The average mortality is about 20 per cent.

Treatment.—The windows of the sick-room should be wide open, regardless of atmospheric conditions. A temperature below 65° F. is preferable. The diet should be fluid or semifluid. Milk, junket, wine-whey, broth, eggs, and gruel are suitable forms of nourishment. Cool water should be given freely. In the absence of any indication for special local treatment the chest may be enveloped in a light woolen jacket.

In robust subjects, at the onset, when the invasion is violent and attended with a bounding pulse, marked dyspnea, and severe pleuritic pain, the abstraction of from 10 to 20 ounces of blood may afford great relief. Later in the course of the disease, if cyanosis and orthopnea develop in consequence of overdistention of the right ventricle, venesection may also prove useful.

Cardiac Weakness.—Alcohol is the best stimulant. When the pulse becomes compressible and the diastolic sound at the pulmonary area loses its force, it should be given freely. The patients who need it most are the aged, the debilitated, and the alcoholic. Digitalis is undoubtedly useful in some cases, but its action is uncertain and often disappointing.

As a circulatory stimulant strychnin usually proves much more efficacious than digitalis. It should be given in ascending doses of from 1/60 to 1/20 grain. In order that there may be immediate absorption, large doses should always be given hypodermically. Caffein is a useful adjuvant to strychnin, but it should not be used when there is marked insomnia. In threatening collapse camphor hypodermically (1 to 2 grains in sterile olive oil every two or three hours) is sometimes very efficient. Atropin is often of value when edema of the lungs is impending. Enemas or subcutaneous injections of normal salt solution are also useful in overcoming adynamia.

Pain.—Morphin hypodermically is the best analgesic. Hot or cold applications are useful. When the pain is very severe, a few wet or dry cups will be found serviceable.

Cough—Hard, dry cough is best relieved by codein (1/8 to 1/4 grain), heroin (1/16 grain), or Dover's powder (3 to 5 grains). Expectorants are rarely needed. If, however, there is much bronchial catarrh, ammonium carbonate may be given to facilitate expectoration.

Fever.—Very high fever is best controlled by the application of ice-bags to the affected side and by cold spongings or cold packs.

Dyspnea.—Cardiac and respiratory stimulants (strychnin, caffein, ammonia) are of service. Oxygen makes the breathing easier, lessens cyanosis, and conduces to sleep, and to this extent aids in conserving energy.

Delirium and Insomnia.—An ice-cap to the head is serviceable. In the early stages of the disease morphin is beneficial and safe. Later, preference should be given to bromids, veronal, or chloralamid.

Tympanites.—Turpentine stupes, turpentine or asafetida enemas, the use of the rectal tube, and hot enteroclysis are the most useful measures.

Delayed Resolution.—Small blisters may be applied over the affected area, and potassium iodid may be administered internally. *X*-rays have proved useful in a few cases.

BRONCHOPNEUMONIA

(Capillary Bronchitis; Catarrhal Pneumonia; Lobular Pneumonia)

Definition.—An inflammation of the terminal bronchioles and air-vesicles.

Etiology.—It is most frequently observed in young children and in the aged. It is a common sequel of the specific fevers, especially of whooping-cough, measles, influenza, and diphtheria. In infants and in debilitated subjects of any age it may occur as a primary affection, the result of exposure.

Another group of cases results from the aspiration of infectious materials or particles of food into the smaller bronchi (aspiration or deglutition pneumonia). This accident is liable to occur whenever the sensibility of the larynx is benumbed, as in apoplexy, bulbar palsy, or uremia. Cancer of the throat and operations on the upper air-passages also favor its occurrence.

The immediate cause is some bacterium. The organism most frequently found is the Diplococcus pneumoniæ. This may occur alone or in combination with the streptococcus, staphylococcus, bacillus of Friedländer, or more rarely with the typhoid bacillus, influenza bacillus, colon bacillus, or diphtheria bacillus.

Pathology.—As a rule, both lungs are involved. On section, small projecting areas of consolidation are noted here and there around the finer bronchioles. Recent patches are reddish-brown in color, firm, and smooth or finely granular; later they become grayish and soft. The terminal bronchi are filled with purulent material.

In addition to these solidified areas there are other small patches of collapsed lung that are airless, firm, and bluish-red in color. The collapse has resulted from occlusion of the bronchus, and closely resembles consolidation; but it can, as a rule, be overcome when inflation is practised by means of a blowpipe inserted in the supplying bronchus.

Microscopic examination reveals an exudate in the terminal bronchi and air-cells, which is composed of leukocytes and desquamated epithelium in various stages of degeneration. The walls of the bronchioles are also infiltrated with leukocytes.

Compared with croupous pneumonia, the contrast is marked. In the latter the lung is involved *en masse;* the consolidation is distinctly granular, and the exudate is composed of red blood-corpuscles, white blood-corpuscles, and fibrin; the lining epithelium is but slightly involved, and the walls of the bronchi are not infiltrated with leukocytes.

Terminations and Sequels.—(1) Resolution; the exudate undergoes autolytic digestion, and is eventually absorbed or expectorated. (2) Death frequently occurs from toxemia and exhaustion. (3) Termination in tuberculosis was supposed formerly to occur very frequently; it is now regarded as being relatively rare. Most of the cases in which this termination is supposed to have occurred were in reality cases of primary tuberculous pneumonia. (4) Pleurisy (fibrinous or purulent) sometimes follows. (5) Abscess and gangrene are common terminations in the aspiration form, but are rare in other types of the disease. (6) Chronic interstitial pneumonia is an occasional sequel.

Symptoms.—The symptoms are often masked by the primary disease. The onset is usually gradual, and is characterized by prostration, cough, and fever. The last is moderately high and very irregular (102°–104° F.). The dyspnea is marked, and the respirations are rapid—50 to 80 a minute; the pulse is greatly accelerated—120 to 160 a minute; cough is painful and accompanied by a mucopurulent expectoration that is occasionally blood-streaked. The face is usually pale and anxious, and the lips blue.

PHYSICAL SIGNS.—As the areas of consolidation are generally small and scattered, the physical signs are inconstant.

Inspection may reveal evidences of dyspnea—lividity, playing of the nostrils, prominence of the sternocleidomastoids, etc.

Palpation usually gives negative results.

Percussion may reveal areas of dulness in one or both lungs.

Auscultation may reveal patches of subcrepitant râles or, perhaps, areas over which the breathing is bronchial or bronchovesicular.

Diagnosis.—*Acute Pulmonary Tuberculosis.*—In this disease there is a tuberculous bronchopneumonia which is difficult to distinguish from simple bronchopneumonia. A family history of tuberculosis, extensive involvement of the apices, bubbling râles indicating softening, the long duration, profuse sweats, rapid emaciation, and the presence of tubercle bacilli and elastic fibers in the sputa are the diagnostic phenomena of tuberculosis.

The following table will show the clinical differences between *bronchopneumonia* and *croupous pneumonia:*

BRONCHOPNEUMONIA	CROUPOUS PNEUMONIA
Often secondary to bronchitis or an acute infectious disease.	Usually a primary disease.
The onset is gradual and without a distinct chill.	The onset is abrupt and with a distinct chill.
The fever is moderately high, very irregular, and ends by lysis after an indefinite period, sometimes of two or three weeks' duration.	The fever is high, regular, and generally ends by crisis between the sixth and ninth day.
The sputum is mucopurulent or glairy and tenacious.	The sputum is rusty and translucent.
Both lungs are commonly affected.	In the majority of cases only one lung is affected.
The physical signs are indistinct and indicate scattered areas of consolidation.	The physical signs are distinct and indicate a large uniform consolidation.

Bronchitis.—In simple bronchitis the fever is not high; the dyspnea is slight, there is little prostration, and there are no signs of consolidation.

Prognosis.—In previously healthy children the prognosis is good. In cachectic children the outlook is very grave. Aspiration pneumonia is usually fatal. The average mortality in childhood is about 35 per cent.

The duration of the disease is from one to three weeks; a longer duration should suggest tuberculosis.

Treatment.—Much can be done by careful management in preventing bronchitis from gaining access to the smaller bronchi.

On the supervention of bronchopneumonia the patient should be confined to bed, in a well-ventilated room, which should be kept preferably at a temperature of about 65° F. The diet should be liquid and nutritious. Alcohol is often required. If the circulatory depression is pronounced, whisky may be given in doses of from 10 to 20 minims in milk to a child of two years every two or three hours.

At the outset it is advantageous to administer a mild purgative, preferably calomel or castor oil. If there is a harsh, dry cough, the application of the tincture of iodin of suitable strength usually affords some relief. In adults sinapisms or stupes may be used instead of the iodin.

Fever is best controlled by cold. Compresses wrung out of cold water may be wrapped around the chest and changed for fresh ones at intervals of twenty minutes. Expectorants are usually required. In the early stage potassium citrate is very serviceable. It may often be combined advantageously with spirit of nitrous ether and ammonium acetate, as in the following formula:

℞.	Potassii citratis	ʒiss
	Spiritus ætheris nitrosi	fʒvj
	Liquoris ammonii acetatis	f℥j
	Syrupi tolutani	
	Aquæ	āā q. s. ad f℥iv.—M.

SIG.—Dessertspoonful every three hours for a child of three years.

Later, the ammonium salts, especially the carbonate, are more efficacious. From 1 to 2 grains of the latter may be given every three or four hours to a child of two years. Ammonium iodid is also useful, and may be employed as an adjuvant, as in the following formula:

℞.	Ammonii carbonatis	gr. xlviij
	Ammonii iodidi	gr. xxiv
	Syrupi tolutani	
	Aquæ	āā q. s. ad f℥iij.—M.

SIG.—Teaspoonful every two or three hours for a child of three years.

If the child is unable to expel the mucus and the breathing becomes much oppressed, an emetic (ipecac or alum) may prove of service. Inhalations of oxygen sometimes make breathing easier. Strychnin is also of benefit at this time in combating respiratory failure.

If symptoms of cardiac failure are pronounced, digitalis must be given in addition to alcohol and strychnin. Extreme restlessness and insomnia will sometimes require the use of the bromids or some other mild sedative.

Except at the onset, when they may be necessary to relieve pleuritic pain and to control harassing cough, opiates should not be used.

Convalescence must be guarded. Tonics like cod-liver oil, iron, and hypophosphites are useful restoratives. A change of air is desirable in protracted cases.

CHRONIC INTERSTITIAL PNEUMONIA

(Cirrhosis of the Lung; Chronic Pneumonia; Pulmonary Induration)

Definition.—A chronic disease of the lung, characterized by an overgrowth of fibrous tissue.

Etiology.—It is rarely a sequel of croupous pneumonia or of the bronchopneumonia. It may be excited by the constant inhalation of irritating dusts, as stone-dust (chalicosis), coal-dust (anthracosis), or metal-dust (siderosis). It may result from syphilis. It is occasionally secondary to chronic pleurisy. It is an invariable accompaniment of chronic tuberculosis.

Pathology.—When the thorax is opened, the lung is found retracted and the heart displaced. The organ is tough, firm, and more or less airless. Section shows an overgrowth of fibrous tissue, and usually inflammation and considerable dilatation of the bronchi.

Symptoms.—The chief symptoms are dyspnea on exertion and cough. The latter may be dry, but it is usually associated with more or less mucopurulent sputum. There is rarely fever, and the general health may be well preserved for many years.

Physical Signs.—*Inspection* reveals retraction of the affected side and displacement of the apex-beat of the heart.

Percussion may yield dulness. Over saccular dilatations of the bronchi there may be a tympanitic note.

Auscultation.—The vocal resonance is increased, and the breathing is often bronchial or cavernous.

Diagnosis.—*Fibroid Phthisis.*—This is often bilateral; fever is a frequent accompaniment, and tubercle bacilli are present in the sputa.

Prognosis.—The disease is incurable. Its course, however, is extremely chronic. Death may be due to intercurrent disease or dilatation of the right heart.

Treatment.—This is largely hygienic and coincides with that laid down for tuberculosis. Stimulant expectorants are useful when bronchitis or bronchiectasis is a prominent feature.

ABSCESS OF THE LUNG

Etiology.—(1) It is rarely a sequel of croupous pneumonia or of bronchopneumonia. (2) It is a frequent accompaniment of tuberculosis. (3) It may be excited by the aspiration of foreign matter into the bronchi. (4) It may result from the extension of a suppurative inflammation in some adjacent part, such as the pleura or liver. (5) Multiple abscesses of embolic origin are of common occurrence in pyemia.

Symptoms.—High and irregular fever, rigors, sweats, pallor, and leukocytosis indicate suppuration. Dyspnea, cough, and purulent, offensive sputa containing shreds of lung tissue are the pulmonary symptoms. Physical examination may reveal bubbling râles, and, later, cavernous breathing and pectoriloquy. Multiple embolic abscesses are rarely recognized during life.

Prognosis.—Many cases following pneumonia and the rupture of external abscesses into the lung end in recovery. Embolic abscess always proves fatal.

Treatment.—Nutritious food and quinin, strychnin, and alcoholic stimulants will be required to support the system. Single abscesses, if they can be localized, should be opened and drained.

GANGRENE OF THE LUNG

Etiology.—Gangrene of the lung is not a primary condition, but is secondary to inflammation or necrosis of the lung tissue. It is excited by the entrance of bacteria of putrefaction, but unless the system is considerably reduced in vitality, the tissues, even though diseased, show wonderful resistance and escape putrefaction.

Pneumonia, especially aspiration-pneumonia, tuberculosis, pressure of morbid growths, bronchiectasis, abscess, and hemorrhagic infarction following embolism of the pulmonary artery are the predisposing pulmonary conditions; Bright's disease, alcoholism, the infectious fevers, and particularly diabetes, by lowering vitality, render the lung more liable to be attacked.

Pathology.—The process may be circumscribed or diffuse. The affected part is converted into a greenish-black, soft mass having an extremely fetid odor. After the softened material has been expectorated, there is left behind a cavity with ragged walls, containing a foul-smelling liquid. The tissues around the cavity are inflamed and edematous.

Symptoms.—Persistent cough, irregular fever, and emaciation are usually present. Hemoptysis is common. The expectoration is characteristic; it is profuse, and has a penetrating, offensive odor. When allowed to stand in a glass vessel, it separates into three layers: a frothy layer on top, a translucent serous layer in the middle, through which hang strings of pus, and at the bottom a layer of reddish-green purulent material. Altered blood may give it the appearance of prune-juice. Microscopically it contains shreds of tissue, crystals of fatty acids, crystals of hematoidin, and numerous pyogenic bacteria.

The *physical signs* are those of solidification followed by those of excavation. Physical signs of pyopneumothorax may supervene from perforation into the pleura.

Prognosis.—This is grave, but not hopeless. Death may result from exhaustion, hemorrhage, or cerebral abscess the result of embolism.

Treatment.—Nutritious food and strychnin, quinin, and alcohol are required to support the system. Inhalations of creosote or of formalin (2 per cent. gradually increased to 5 per cent.) may be employed to lessen the fetor of the breath. Surgical interference is indicated if the gangrenous process can be localized and is not a complication of an incurable disease.

PULMONARY TUBERCULOSIS

(Phthisis; Pulmonary Consumption)

Definition.—A specific inflammatory disease of the lungs caused by the Bacillus tuberculosis.

Etiology.—The disease most commonly develops between the ages of fifteen and forty. Though tuberculosis is very rarely transmitted from parent to offspring, an inherited susceptibility to the disease is not rarely observed. Overcrowding, bad ventilation, lack of sunlight, and poor food; occupations that necessitate the breathing of impure air and irritating dusts; a small, flat chest; and certain other diseases, such as catarrh of the respiratory tract, whooping-cough, measles, diabetes, and cirrhosis of the liver, favor infection.

Infection may take place: (1) By the inhalation of air laden with moist particles of infected sputum, expelled in coughing, sneezing, etc. (droplet infection), or with the dust of dried tuberculous sputum; (2) by the ingestion of food contaminated directly or indirectly with infected sputum, of the milk, or rarely the meat, of tuberculous cattle, or of bacilli-infected material that has been conveyed to the mouth by the fingers, drinking-cups, toys, etc.; (3) by the direct ino lation of wounds (rare); (4) by direct parental (placental) transmission (very rare).

The bacilli may reach the lungs directly through the air-passages, or they may be brought to these organs from the intestines, tonsil, or some other portal of entry by the lymphatics or blood-vessels.

Pathology.—The exciting cause is the *Bacillus tuberculosis*, a minute rod, about one-fourth or one-half as long as the diameter of a red blood-cell, and often slightly bent and beaded. Its detection depends on the power of the stained bacillus to resist the decolorizing effects of acids. Several types of bacilli exist, but only two, the human and the bovine, appear to be infectious for human beings. The bovine organism, which is distributed chiefly by means of milk, is not an uncommon cause of tuberculosis in young children, but only a very small proportion of cases in adults are due to this type.

The lodgment of the bacilli in the terminal bronchioles or peribronchial tissues excites a proliferation of the fixed connective-tissue cells. The new cells, from their resemblance to epithelial cells, are known as *epithelioid cells.* They have a relatively large amount of protoplasm and a rather faintly staining vesicular nucleus. *Giant-cells* are sometimes formed by the fusion or overgrowth of the epithelioid cells. In consequence of the local irritation the focus of epithelioid cells is soon surrounded by a wall of *small round cells* (lymphocytes and proliferated fixed connective-tissue cells), the whole forming a gray, translucent mass—the gray tubercle of Laennec. In a short time the tubercle toxin brings about necrotic changes in the cellular accumulation, and these gradually transform the tubercle into a yellow, cheesy mass—the yellow tubercle of Laennec. The degenerated tubercles fuse and form the uniform cheesy masses so commonly observed at the autopsy. At this stage one of two things may occur: the mass may soften, discharge through a bronchial tube, and leave behind a cavity with ulcerating walls, or it may become encapsulated by an overgrowth of connective tissue and subsequently calcified.

The destructive changes are usually due to the association of pyogenic cocci with the tubercle bacilli. In some very acute cases of tuberculosis the infiltration is not in the form of definite tubercles, but is diffuse, the entire mass at first presenting a peculiar grayish-red gelatinous appearance, and later becoming distinctly caseous.

In addition to the specific lesions other secondary changes are noted. The lung tissue adjacent to the tuberculous deposits is often the seat of a true pneumonic inflammation; the connective tissue is almost always more or less increased; the bronchi are inflamed; and the pleural surfaces over the affected areas are frequently adherent.

Forms.—Two chief forms of pulmonary tuberculosis are recognized: The acute and the chronic. Acute tuberculosis occurs in three subtypes. In the *acute penumonic* type a large area of the lung is uniformly infiltrated as in croupous pneumonia, and the process rapidly proceeds to caseation and softening. In the *bronchopneumonic* type the infiltration occurs in more or less discrete patches surrounding the final distributions of the bronchi. By fusion of the foci a lobe or even an entire lung may ultimately become involved. In the *acute miliary* type the lungs are studded with countless discrete tubercles of the size of a millet seed or a little larger. This condition which is often secondary to chronic tuberculosis of the lung, is usually a part of widespread infection involving many organs (general miliary tuberculosis), dissemination of the bacilli having taken place through the blood stream.

Chronic ulcerative tuberculosis, the most common form of pulmonary tuberculosis, begins with the development of gray tubercles, usually in the apical region, and tends to spread downward, various forms of lesions—cavity, caseation, conglomerate tubercles, discrete fresh tubercles—often being found at autopsy in the same lung. In many cases, howeve the infection is arrested at an early stage, and in this event th only lesion at death may be a small cheesy or calcareous mass surrounded or pervaded by fibrous connective tissue. In some instances the term *chronic fibroid tuberculosis* is applicable, as the progress of the disease is held in abeyance for years by an enormous hyperplasia of connective tissue surrounding and isolating the tuberculous areas. Bronchiectasis and pleuritic adhesions are always present.

Symptoms of Acute Pneumonic Tuberculosis.—Clinically this form in the beginning closely resembles croupous pneumonia or bronchopneumonia, but the symptoms instead of subsiding at the end of ten days or two weeks persist and gradually become more or less characteristic. The fever assumes a remittent or an intermittent type, chills and sweats occur, signs of softening and excavation develop in the affected lung, anemia and emaciation become pronounced, and death results in from four to eight weeks; or, more rarely, a certain degree of improvement occurs and the clinical picture slowly changes to that of chronic ulcerative tuberculosis.

Acute Miliary Tuberculosis.—The symptoms of acute miliary tuberculosis are described on page 383.

Symptoms of Chronic Ulcerative Tuberculosis.—The onset is usually insidious and marked by pallor, gastric disturbance, loss of flesh and strength, and by a dry, hacking cough that is noted especially in the morning. From some undue exposure the cough is often aggravated, and to this obstinate "cold" the disease is usually attributed. In some cases the symptoms appear abruptly with hemorrhage or an acute pleurisy. Occasionally the first manifestation is gradually developing hoarseness.

Slight fever and acceleration of the pulse are early symptoms of great diagnostic import. The temperature is marked by an evening exacerbation, during which the face is flushed, the eyes are bright, and the mind animated. As the disease advances the cough becomes troublesome and the expectoration more abundant. In well-developed cases the expectoration is greenish-gray in color, is in coin-shaped plugs (nummular), is heavy and sinks in water, is often blood-streaked, and on microscopic examination is found to contain bacilli and fibers of elastic tissue.

Pleuritic pains are often present. Hemoptysis occurs in from 50 to 60 per cent. of all cases. In the later stages the bleeding is sometimes profuse, although it is not often immediately fatal. Vomiting, the result of the cough or of secondary gastric disease, is common. The respiration is accelerated, but patients rarely complain of dyspnea except upon exertion.

The final stage is characterized by extreme emaciation, weakness, pallor, remittent or intermittent fever of a hectic or septic type, and sometimes edema of the feet. The mind is usually clear and peculiarly hopeful to the end.

PHYSICAL SIGNS.—The chest may be well formed. Often, however, it is long and flat, with hollow supraclavicular and infraclavicular spaces, prominent scapulæ, and oblique ribs. When the disease is well advanced, there may be retraction with diminished expansion over one apex. Slender tapering fingers with incurving of the nails, or, on the other hand, short broad finger-tips ("clubbed" fingers) are often noted.

Palpation.—This reveals imperfect expansion and exaggerated vocal fremitus.

Percussion.—Dulness over the solidified areas can be detected at an early period of the disease. It may be obtained first above or below the clavicles, in the supraspinous fossæ, between the scapulæ, or in front, near the sternal border.

Cavities unless they are filled with fluid, yield a typanitic sound, or if thin-walled, a so-called cracked-pot note. The latter is best obtained with quick, light percussion strokes, when the patient's mouth is open.

Auscultation.—In the earliest stage the respiratory sounds may be feeble over the affected area. Later the breathing is harsh and the expiration is prolonged (bronchial). The vocal resonance is increased. Crackling râles are usually audible, especially after coughing. Auscultation over cavities may reveal cavernous or amphoric breathing, bronchophony or pectoriloquy, and large gurgling râles.

Anomalous Physical Signs.—The vocal fremitus is diminished when there is much pleural thickening. Normal resonance or hyperresonance may replace dulness when there is much emphysema between small tuberculous foci. Weak breathing may replace bronchial or cavernous when the tubes or cavity are filled with mucopus. The signs of cavity are sometimes produced by consolidation in the neighborhood of a large bronchus.

Chronic Fibroid Tuberculosis.—This form may last many years. It is characterized by gradual loss of flesh and strength, dyspnea, and mucopurulent expectoration which is at times fetid from retention in bronchiectatic cavities. Fever and sweating are usually slight. Hemoptysis is somewhat frequent. Clubbing of the fingers is often marked. The physical signs are much like those of chronic ulcerative tuberculosis, but retraction and deformity of the chest-wall is a more conspicuous feature.

Complications.—The chief complications of pulmonary tuberculosis are hemoptysis, bronchopneumonia, pleurisy, pneumothorax or pneumopyothorax, gastro-intestinal catarrh, rectal fistula, amyloid degeneration of the viscera, tuberculosis of other structures (larynx, meninges, intestines, kidneys, peritoneum, etc.), and general miliary tuberculosis.

Diagnosis.—Slight fever, especially toward evening, a frequent irritable pulse, persistent cough, progressive loss of weight, hemoptysis, signs of infiltration of the lung and the occurrence of so-called idiopathic pleurisy are suggestive features. The various tuberculin tests may be employed in doubtful cases, but their value should not be overestimated as they are not uncommonly positive in apparently normal individuals. The presence of tubercle bacilli or of elastic fibers in the sputum proves the existence of tuberculosis, but it is a comparatively late indication.

Prognosis.—In acute tuberculosis the outlook is unfavorable. In chronic tuberculosis the prognosis is dependent upon the stage of the disease, the constitutional vigor of the subject, and the hygienic conditions under which he is obliged to live. The mortality is high in young subjects (fifteen to twenty-five years) and those of feeble constitution. Unfavorable prognostic signs are persistent high temperature, acceleration of the pulse out of proportion to the fever, involvement of both lungs, continued indigestion, progressive loss of flesh, and the development of tuberculous lesions in other organs. The average duration is about three years.

Treatment.—*Prophylaxis.*—The means that may be instituted by the public authorities to limit the spread of tuberculosis include: The dissemination of information concerning the prevention of the disease; the supervision of schools, tenement-houses, factories, public conveyances, etc.; the systematic inspection by skilled veterinarians of dairies and slaughter-houses with the view of declaring unmarketable the milk and meat of tuberculous animals; the suppression of promiscuous expectoration in public places; the establishment of special hospitals and dispensaries for the indigent suffering from tuberculosis; and the compulsory registration of phthisical patients. Tuberculous patients should be taught to expectorate only into proper receptacles containing a disinfectant solution (5 per cent. carbolic acid) or into moistened rags or paper napkins, that should be burned before the sputum becomes dry. They should sleep alone. Their rooms should be sunny, well ventilated, and kept scrupulously clean.

Persons with a predisposition to tuberculosis can do much to increase their powers of resistance by strict attention to hygiene. Fresh air and sunlight, a healthy residence, an outdoor occupation, the wearing of warm clothes, with flannel next to the skin, and a diet of wholesome and nutritious food, temperate living, systematic exercise, and daily cold sponging, followed by friction of the skin, are the factors to be relied upon in attempting to overcome individual susceptibility.

Sanatorium treatment undoubtedly gives the patient the best chance of recovery. In such institutions the patient spends in summer not less than nine or ten hours, and in winter not less than from six to nine hours, in the open air. The bedroom windows are kept open both winter and summer. He is given a mixed diet of wholesome food, and encouraged to eat as heartily as his digestive capacity will permit. If the disease is active, he is kept at absolute rest. For the most of the day he lies on a bamboo couch in the open air, warmth being maintained by abundant covering and, if necessary, by a hot stone at the feet. In quiescent tuberculosis moderate exercise is recommended, every precaution being taken, however, to guard against fatigue. To secure lasting improvement, the patient should remain in the sanatorium at least six months.

Climatic Treatment.—To patients to whom a protracted stay in a sanatorium would be irksome or distasteful a change of climate offers the greatest hope of cure. As a rule, a high altitude should be selected; the atmosphere should be dry, and the temperature equable. Personal experience must decide the question of temperature; generally patients who feel better in summer will do well in a warm climate, and *vice versa.* The physician should have some knowledge of the locality, which should afford ordinary conveniences without being too crowded with sufferers similarly afflicted.

In selected cases a sea voyage is often very useful. According to Douglas Powell, it is most suitable to patients in the early stages, who have been previously healthy, who have overworked nervous systems, and in whom the disease is more or less quiescent.

Treatment at Home.—This should be made to imitate as closely as circumstances will permit that which is followed in the sanatorium. The airiest and sunniest room should be selected for the patient. So long as he has fever absolute appears to be effective in chronic cases. A small amount of an ointment of europhen (10 per cent.), a compound containing much loosely combined iodin, may be rubbed into the chest twice daily.

Symptomatic Treatment.—*Cough.*—In many cases cough is indispensable and is best treated by promoting expectoration. For this purpose creosote, guaiacol carbonate, terebene, oil of eucalyptus, and myrtol are reliable remedies. Inhalations of creosote, compound tincture of benzoin, or terebene are often very effective. Local blistering is also of service. If the cough is very severe, sedatives must be given. Of these, the least objectionable are codein, heroin, hydrocyanic acid, and spirit of chloroform. Such combinations as the following will be found useful:

℞. Codeinæ sulphatis	gr. vj-viij
Spiritus chloroformi	fʒj
Glycerin	f℥j
Succi limonis	f℥ss
Aquæ	q. s. ad f℥iij—M.

Sig.—A teaspoonful as occasion demands.

℞. Codeinæ sulphatis	gr. iv
Acidi hydrocyanici diluti	♏ xxxij
Syrupi tolutani	q. s. ad f℥ij.—M.

Sig.—A teaspoonful as required.

Night-sweats.—Sponging the body at bedtime with a solution of alum in alcohol and water or dusting it with a powder of tannoform (1 part) and zinc oxid (3 parts) is sometimes very effective. The most reliable internal remedies are atropin (1/200 to 1/120 grain), picrotoxin (1/80 to 1/40 grain), and camphoric acid (5 to 10 grains).

Pyrexia.—In many cases the fever yields to absolute rest in bed or in a reclining chair, combined with life in the open air. Cold sponging is useful if temperature is high. In obstinate cases the administration of phenacetin (3 to 5 grains) may be tried.

DISEASES OF THE PLEURA

PLEURISY

(Pleuritis)

Definition.—Inflammation of the pleura.

Varieties.—According to cause it may be divided into primary or secondary; according to extent, into unilateral, bilateral, or localized; according to duration, into acute or chronic; and according to the exudation, into serofibrinous, fibrinous, or purulent.

Etiology.—Pleurisy may be: (1) Idiopathic, arising from exposure to cold and wet; (2) secondary to inflammatory diseases of adjacent structures, such as pneumonia, pulmonary tuberculosis, etc.; (3) secondary to general morbid processes that lessen tissue resistance, such as the specific fevers, chronic nephritis, cancer, etc.; (4) traumatic; (5) tuberculosis. *At least three-fourths of all cases of so-called idiopathic pleurisy are tuberculous, and in at least one-fourth of such cases definite signs of pulmonary involvement sooner or later develop.* The bacteria most commonly found in the exudate are the tubercle bacillus, pneumococcus, and streptococcus.

Pathology.—In the early stage the membrane is red, sticky, lusterless, and covered with a thin film of fibrin; if the process now ceases, the condition is termed *dry pleurisy*. If, however, the inflammation continues, an exudate is formed which may be: (1) Serofibrinous; (2) fibrinous; or (3) purulent (empyema). In the *serofibrinous* form there is little fibrin, the exudate being mainly composed of straw-colored serum (a few ounces to several pints), which in favorable cases is pain. After the development of a liquid effusion the characteristic features are immobility, bulging of the intercostal spaces, and displacement of the apex-beat of the heart.

Palpation reveals immobility of the affected side and absence of the vocal fremitus.

Percussion yields marked dulness or flatness and a sensation of increased resistance. The upper line of dulness is not horizontal, but is curved and rises higher posteriorly. In moderate effusions the level of dulness often changes with the position of the patient. Above the effusion percussion gives a tympanitic note (Skoda's resonance). With the patient in the erect posture a triangular patch of dulness can usually be detected along the spine at the base of the unaffected side (*Grocco's sign*). In left-sided effusions Traube's semilunar space is obliterated.

Auscultation.—In the early stage this detects a to-and-fro friction-sound of respiratory rhythm. After the development of the effusion the respiratory sounds are weak and distant. Occasionally they have a tubular quality, especially near the margin of the liquid. Vocal resonance is usually diminished or absent, but occasionally, if the effusion is moderate, egophony may be heard. The friction-sound may again be audible when the fluid disappears.

Mensuration shows an increase in the size (½ to 1 inch) of the affected side.

Cytodiagnosis.—A preponderance of lymphocytes in the exudate suggests chronic tuberculous pleurisy, while a great excess of polynuclear leukocytes points to pneumococcic or streptococcic infection.

Diagnosis.—*Croupous Pneumonia.*—The severe chill, rusty expectoration, high fever, the fine inspiratory râles, dulness not changing with the patient's posture, increased vocal fremitus, increased vocal resonance, loud bronchial breathing, and the absence of bulging and of displaced apex-beat will serve to distinguish pneumonia from pleurisy.

Pleurodynia.—In this affection the pain and tenderness are diffuse; moreover, fever, friction-sounds, and signs of effusion are absent.

Diaphragmatic Pleurisy.—This may present the following symptoms: Intense pain under the margin of the ribs, with tenderness on pressure; thoracic breathing; tenderness over the phrenic nerve, which is accessible between the two roots of the sternocleidomastoid at the base of the neck; hiccup; and severe dyspnea. The physical signs are not marked.

Pericarditis with Effusion.—In this condition the area of dulness has a characteristic shape, the sounds of the heart are distant and muffled, and there is greater embarrassment of the circulation.

Hydrothorax.—In this condition pain and fever are absent. There is often a history of cardiac or renal disease, and the fluid on aspiration is found to contain less than 3 per cent. of albumin and to have a specific gravity below 1015.

Pyothorax.—This may be recognized by the general symptoms of sepsis—persistent irregular fever, increasing pallor, profuse sweats, chills, and leukocytosis. In doubtful cases it will be necessary to resort to the exploratory needle.

Prognosis.—In simple serofibrinous pleurisy the prognosis is guardedly favorable. Fever usually subsides in from a week to ten days, and absorption of the fluid in many cases is complete in from four to six weeks. In other cases (usually tuberculous) the effusion remains for months or recurs again and again after aspiration. Sudden death occasionally occurs when the fluid is excessive. In about one-fourth of the cases signs of pulmonary tuberculosis sooner or later develop.

Treatment.—The patient should be kept in bed and restricted to a liquid or semiliquid diet. Mercurial or saline aperients may be prescribed at the onset. For the severe pain the application of a blister or of wet or dry cups, together with the administration of morphin, will be found effective. Strapping the affected side with broad strips of adhesive plaster is also useful. Acute sthenic cases with decided fever are often favorably influenced by the administration of salicylates (1 to 1½ drams of the sodium or ammonium salt a day). In asthenic cases salicylates are of no avail.

Removal of Serous Effusion.—The most useful measures for promoting absorption are the application of iodin or of flying blisters, and the administration of hydragogue cathartics and of diuretics. From ½ to 1 ounce of magnesium sulphate may be given in as little water as possible an hour before breakfast, and the fluid consumed by the patient during the day restricted to a minimum. The most serviceable diuretics are digitalis, caffein, theobromin, and potassium acetate. Potassium iodid (5 to 10 grains thrice daily) is also employed for its absorbent effect. Diaphoretics are of no value.

Paracentesis is demanded—(1) if the effusion is considerable and shows no signs of receding after the lapse of two weeks; (2) if there is sufficient fluid to cause severe dyspnea, cyanosis, persistent cough, or failing pulse; (3) if the fluid reaches the level of the second rib and there is marked dislocation of the adjacent organs; (4) if the presence of pus is suspected.

The most favorable site for the puncture is usually in the sixth or seventh intercostal space, between the mid-axillary line and the angle of the scapula. After anesthetizing the part, the needle should be introduced with a quick stroke along the upper margin of the rib. The fluid should be removed slowly, and under no circumstance should extreme efforts be made to obtain the largest possible amount. The operation should be terminated at once if incessant cough, severe pain, dyspnea, palpitation, tendency to syncope, or other untoward symptoms appear.

Purulent Pleurisy (Empyema, Pyothorax).—The effusion may be primarily purulent, having been excited by pyogenic micro-organisms, or a serofibrinous effusion, through subsequent infection, may become purulent. Traumatism or the rupture of a purulent accumulation into the pleural sac is an occasional cause. It frequently follows pneumonia, particularly in children, in whom the most common form of pleurisy is empyema. It is sometimes secondary to tuberculosis or one of the infectious fevers.

The organisms most frequently present are the pneumococcus, streptococcus, staphylococcus, and tubercle bacillus.

Symptoms.—The physical signs and symptoms are similar to those observed in serofibrinous pleurisy. Pus is indicated by septic phenomena—high and irregular fever, sweats, chills, pallor, and leukocytosis; by the results of aspiration; and sometimes by edema of the chest-walls. In pulsating pleurisy the effusion is almost always purulent.

Prognosis.—Grave, though recovery frequently occurs. The most favorable cases are those following pneumonia.

Treatment.—This consists in free incision and thorough drainage. Irrigation is unnecessary unless the fluid is putrid. In long-standing cases the excision of several ribs (Estlander's operation) facilitates retraction and the obliteration of the pleural sac, which is essential to a cure.

HYDROTHORAX

Definition.—An accumulation of serum of non-inflammatory origin in the pleural sac. The condition may be unilateral or bilateral.

Etiology.—It may result from the usual causes of edema—nephritis, chronic heart disease, emphysema, anemia, etc., it may be due to pressure on the azygos or pulmonary veins by a tumor, aneurysm, or dilated right auricle. In cardiac disease it is usually unilateral and on the right side, or if bilateral more marked on the right side.

Symptoms.—It gives rise to dyspnea, cyanosis, and the physical signs of a pleural effusion.

Diagnosis.—This is based upon the history, the absence of pain and fever, and the character of fluid obtained by aspiration (see p. 311).

Treatment.—Remedies should be directed to the primary disease. If hydragogue cathartics and diuretics fail to afford relief and the dyspnea becomes urgent, aspiration must be practised.

PNEUMOTHORAX

Definition.—The presence of air or gas in the pleural sac.

Etiology.—About 90 per cent. of the cases result from the rupture of a tuberculous cavity into the pleura. Less common causes are rupture of the lung from abscess, gangrene, emphysema, etc.; rupture into the lung of an empyema; penetrating wounds of the chest (including thoracentesis); and perforation of the pleura in ulcer or cancer of the esophagus or stomach. Very rarely gas is produced spontaneously in a pleuritic exudate by the gas bacillus (Bacillus aërogenes capsulatus).

Pathology.—The adjacent viscera are often much displaced and the lung is compressed. Even when air alone has escaped into the pleural sac, an effusion soon forms, so that *pneumohydrothorax* or *pneumopyothorax* is an almost inevitable result.

Symptoms.—The onset is frequently characterized by localized pain, marked dyspnea, cough, fall in temperature, feeble pulse, and even a condition of collapse. If, however, the air is confined by adhesions or effusion to a small area (circumscribed pneumothorax) no special symptoms may arise.

PHYSICAL SIGNS.—*Inspection* may reveal distention of the affected side, immobility, and marked displacement of the apex-beat of the heart.

Palpation.—Vocal fremitus is diminished (or absent).

Percussion.—The note is usually tympanitic, but it may be dull if the sac is very tense. At the base there may be flatness, changing with the posture of the patient.

Auscultation.—The respiratory murmur and vocal resonance are usually absent, but if the opening in the lung remains patulous, amphoric breathing may be detected. Metallic tinkling is often heard. When a silver coin is placed on the affected side and is struck with another, the auscultator may detect a clear metallic sound (bell-tympany). If fluid is present, shaking the patient elicits a splashing sound (Hippocratic succussion).

Diagnosis.—*A Large Tuberculous Cavity.*—This is usually located near the apex instead of the base; the surface is sunken, not prominent; the heart is not displaced; succussion-splash and bell-tympany are rarely obtainable.

Dilated Stomach.—This may give a tympanitic note over the left pulmonary base, and may simulate a pneumothorax; but the tympanitic note is continued down into the abdomen, and the swallowing of liquid is distinctly audible over the base of the chest.

Prognosis.—This depends on the cause. In tuberculous subjects it usually proves fatal in from a few days to a few months. In empyemic and traumatic cases the outlook is distinctly more favorable.

Treatment.—In tuberculous cases the indications are to relieve distress by morphin and to combat collapse by such stimulants as ether, ammonia, camphor, alcohol, and strychnin. Aspiration occasionally affords temporary relief. In non-tuberculous cases of pneumopyothorax operative interference is usually advisable.

HEMOTHORAX

Definition.—Blood in the pleural cavity.

Etiology.—It usually results from wounds of the chest-wall, fracture of the ribs, or the rupture of an aneurysm. A sanguineous inflammatory (*hemorrhagic pleurisy*) exudate frequently occurs in cancerous and tuberculous pleurisy and in simple pleurisy if the individual is profoundly anemic.

Symptoms.—The symptoms and physical signs are those of pleural effusion.

ACUTE INFECTIOUS DISEASES

FEVER

FEVER is an abnormal condition, characterized by elevated temperature, quickened respiration and circulation, faulty secretions, and increased tissue-waste. It is dependent upon a perversion of the physiologic processes whereby the generation and the loss of heat are so balanced as to maintain the normal temperature.

The Detection of Fever.—There is only one reliable way of detecting fever, and that is by means of the clinical thermometer. The instrument may be placed in the mouth, axilla, rectum, or vagina.

When the mouth is selected, the bulb should be placed under the tongue and the lips kept closed. Hot or cold drinks recently taken mar the result. For obvious reasons the mouth should not be used in delirious patients.

When the axilla is selected, the following precautions must be observed: Wipe off the perspiration and dry the skin; insert the bulb of the instrument deep in the armpit, and see that the arm is kept close to the side. The thermometer should be kept in position until the mercury maintains the same level for two minutes; this will usually require in all about six or seven minutes.

The rectum may be selected in children. The rectal temperature is slightly above that of the mouth and from ½° to 1° F. above that of the axilla.

Febrile Stages.—The course of all fevers is marked by three stages: (1) Invasion; (2) fastigium, or stadium; (3) defervescence, or decline.

Invasion.—During this period the temperature gradually rises until it reaches its maximum.

Fastigium.—In this period, though there may be marked variations, the temperature shows a tendency to touch again and again its highest point.

Defervescence.—In this period the temperature gradually falls until it reaches the norm.

Terminations of Fever.—Fever terminates by lysis or crisis.

Lysis.—The temperature falls slowly by slight gradations until it reaches the norm.

Crisis.—The temperature falls suddenly—often four or five degrees in twelve or twenty-four hours. In croupous pneumonia, typhus fever, erysipelas, relapsing fever, and malarial fever the fever usually ends by crisis.

The Degree of Pyrexia.—The following is Wunderlich's classification of febrile temperatures:

1. Subfebrile, temperature 99.5°–100.4° F.
2. Slightly febrile, temperature 100.4°–101.3° F.
3. Moderately febrile, temperature 101.3°–103.1° F.
4. Decidedly febrile, temperature 103.1°–104° F.
5. Highly febrile, temperature above 103.1° F. in the morning and above 104.9° F. in the evening.
6. Hyperpyretic, temperature above 106° F.

Febrile Remissions.—All fevers show diurnal variations. The maximum temperature is usually reached at about 6 P. M. and the minimum at about 6 A. M. Occasionally these extremes are reversed and the maximum is in the morning and the minimum in the evening. The daily difference usually amounts to about 1° F.

Types of Fever.—According to the degree of the diurnal variation three types are recognized:

1. *Continued Fever.*—The diurnal variation is slight—1°–1.5° F. Typhus fever, pneumonia, and scarlet fever are examples of continued fevers.

2. *Remittent Fever.*—The diurnal variation is marked, but

the minimum temperature is still above the norm. Typhoid fever, remittent fever, and septic fever are examples of this type.

3. *Intermittent or Interrupted Fever.*—The diurnal variation is marked, and the minimum is normal or subnormal. The following fevers show multiple intermissions:

1. Intermittent malarial fever.
2. Relapsing fever.
3. Malta fever (the febrile periods usually last from one to three weeks and the afebrile periods, few days).
4. Septicemic fever (this may be intermittent or remittent).
5. Hepatic intermittent fever (see p. 100).

A *single intermission or marked remission* is observed in the following fevers:

Smallpox (a remission occurs about the third day).

Yellow fever (a remission usually occurs about the third or fourth day).

Measles (a distinct remission often occurs on the second or third day).

Dengue (a decided remission occurs on the third or fourth day and lasts about a day).

Causes of Fever.—The chief cause is disturbance of the heart-regulating centers by toxic substances circulating in the blood. These substances may owe their origin to bacterial invasion (acute infectious diseases), to faulty metabolism (acute gout, thermic fever), or to mechanical, thermic, or chemical injury of the tissues. Occasionally, as in hysteria, fever appears to be due to a direct disturbance of the heat-regulating centers.

Symptoms of Fever.—The temperature is elevated, the pulse is accelerated, the respirations are increased, the tongue is coated, the appetite is impaired, the secretions of the alimentary canal are deficient, and the urine is scanty, dark colored, and of high specific gravity. Persistent fever is attended with more or less wasting of the body.

The pulse-temperature ratio:

A temperature of 98.4° F. corresponds to a pulse of 70.
A temperature of 100° F. corresponds to a pulse of 80–90.
A temperature of 102° F. corresponds to a pulse of 100–110.
A temperature of 104° F. corresponds to a pulse of 120–130.

Effects of Fever on the Tissues.—High and long-continued fever induces certain marked changes in the tissues, especially cloudy swelling, fatty degeneration, and coagulation necrosis.

Period of Incubation.—The period elapsing between the occurrence of the infection and the development of symptoms. It varies considerably in the same disease, being more or less influenced by the susceptibility of the patient and the virulence of the contagion. The average period of incubation in the various fevers is as follows:

Typhoid fever: five to twenty-one days.
Typhus fever: a few days to two weeks.
Measles: seven days to two weeks.
Rötheln or rubella: five days to three weeks.
Scarlatina: three to seven days.
Smallpox: ten days to two weeks.
Erysipelas: three to seven days.
Diphtheria: two to seven days.
Varicella: fourteen to sixteen days.
Tetanus: a few days to three weeks.
Mumps: two to three weeks.
Yellow fever: from three to four days.
Cholera: two to five days.

The date at which rashes appear in the various acute infections:

Typhoid fever: seventh to the ninth day.
Typhus fever: fourth or fifth day.
Smallpox: third or fourth day.
fourth day.
l day.

Rötheln or rubella: first or second day.
Varicella: first day.

Protection from Future Attacks.—Few diseases confer absolute immunity against future attacks, but the following are fairly protective:

Typhoid fever: relapses are common, but second attacks are infrequent.

Typhus fever: second attacks are very rare.
Measles: second attacks are uncommon.
Rubella: second attacks are rare.
Scarlet fever: second attacks are rare.
Smallpox: second attacks occasionally occur.
Mumps: second attacks are rare.
Varicella: second attacks are uncommon.
Yellow fever: second attacks are rare.
Cerebrospinal fever: second attacks have been occasionally observed.

The following specific fevers do not confer immunity:

Erysipelas.
Relapsing fever.
Diphtheria.
Rheumatic fever.
Malarial fever.
Influenza.
Croupous pneumonia.

Protracted Cases of Fever.—Fever of a remittent or intermittent type lasting from a few weeks to two months or longer, and unaccompanied by very definite physical signs may occur in: (1) Typhoid fever; (2) malarial fever (estivo-autumnal infection); (3) tuberculosis; (4) septicemia (in particular subacute or chronic infectious endocarditis) (5) infection of the urinary passages by the colon bacillus; (6) certain diseases of the blood or hemopoietic system, such as pernicious anemia, leukemia and Hodgkin's disease; (7) secondary syphilis; (8) chronic cerebrospinal fever; (9) malta fever; (10) hysteria (rare).

Infections in which jaundice is likely to occur:

Yellow fever.
Relapsing fever.
Acute yellow atrophy of the liver.
Remittent malarial fever.

SUBNORMAL TEMPERATURE

Temperatures below 97.5° F. may be considered subnormal They are observed in the following conditions:

1. During convalescence from certain febrile diseases. After pneumonia and typhoid fever the temperature may remain subnormal for several days.
2. In collapse from various causes.
3. In cholera. In this disease the temperature may be very low (90°–85° F.) for several days.
4. In certain chronic diseases, especially myxedema, diabetes, cancer, chronic cardiac, cerebral, and spinal diseases

FEBRICULA

(Simple Continued Fever; Ephemeral Fever)

Definition.—An acute febrile disease, of short duration, without definite lesions or a specific etiology.

Etiology.—It is generally met with in young and sensitive individuals. Exposure to the sun, prolonged physical or emotional excitement, and errors in diet seem to excite it.

Symptoms.—The disease usually begins abruptly with chilliness, headache, malaise, and fever which soon attains a maximum of 102° or 103° F. The face is flushed; the pulse is full and rapid; the urine is scanty and high colored; the tongue is coated; the appetite is lost; and the bowels are constipated. There is no characteristic eruption, but herpes is frequently observed on the lips.

The disease lasts from a few days to two weeks, and may end by crisis or lysis.

Diagnosis.—Care must be taken to exclude local inflammations, such as tonsillitis and otitis media, and other fevers.

Typhoid Fever.—At first the diagnosis may be impossible, but the absence of diarrhea, tympanites, abdominal tenderness, splenic enlargement, Widal reaction, and eruption will soon make the diagnosis apparent.

Estivo-autumnal Fever.—The history, the splenic enlargement, and the presence of hematozoa in the blood will serve to distinguish this disease from simple continued fever.

Prognosis.—Favorable.

Treatment.—The patient should be confined to bed and placed upon a liquid or semiliquid diet. Fractional doses of calomel may be employed to relieve constipation.

The fever is best controlled by sponging with water and alcohol and by the use of some mild refrigerant mixture like the following:

℞.	Tincturæ aconiti	♏xxiv
	Spiritus ætheris nitrosi	fʒv
	Liquoris ammonii acetatis	q. s. ad f℥iij.—M.

SIG.—A dessertspoonful every two hours for a child of four years.

TYPHOID FEVER

(Enteric Fever; Typhus Abdominalis)

Definition.—An acute infectious disease, excited by the *Bacillus typhosus,* characterized anatomically by definite lesions in Peyer's patches, mesenteric glands, and spleen; and manifested clinically by fever, headache, delirium, abdominal distention and tenderness, diarrhea, enlargement of the spleen, and a rose-colored rash.

Etiology.—Typhoid fever occurs at all ages, but it is especially frequent in the second and third decades. It appears in all seasons, but it is most prevalent in the late summer and in autumn. Defective drainage and unsanitary conditions in general act as predisposing factors. Certain persons and certain families seem to be more liable to infection than others. One attack usually, but not invariably, confers permanent immunity.

The feces and urine of the patient are the sources of infection, and drinking water contaminated with them is the chief medium of transmission. Milk contaminated after leaving the cow is another important medium of infection. Many cases have been traced to articles of food eaten raw, such as oysters fattened in befouled water, or celery, lettuce, etc., grown in polluted soil. Flies may be an important agent in disseminating the disease. Persons closely associated with typhoid fever patients, such as nurses and attendants are sometimes infected directly by the stools or urine. Small outbreaks are often traceable to so-called "carriers," persons who though perfectly healthy constantly have typhoid bacilli in their stools. It is probable that in rare instances infection occurs by way of the respiratory tract.

Pathology.—The characteristic lesions are found in the abdominal lymphatics, namely, in Peyer's patches, solitary glands, and mesenteric glands. The changes in Peyer's glands are best studied in the lower part of the ileum, which should be opened on the side of the mesenteric attachment.

In the first few days the glands are swollen and hyperemic; later there is a marked cell-proliferation, the blood-vessels are compressed, and the glands become pale and prominent (medullary infiltration). If the disease advances, necrosis sets in, the glands becoming yellow and soft. In a few days the necrotic tissue is discharged, leaving an oval, ulcerated surface with somewhat irregular margins, and a smooth base formed by the submucous coat, muscular coat, or peritoneum. In the fourth week cicatrization begins, and the gland is ultimately replaced by a smooth depressed scar.

In mild cases the stage of ulceration may not be reached, the proliferated cells being removed by fatty degeneration and absorption without rupture of the gland. The solitary and mesenteric glands pass through similar changes, but the latter rarely rupture. Other lesions are found that are not characteristic. The mucous membrane of both the small and large intestines shows catarrhal changes. The spleen is soft and swollen. The liver, kidneys, and heart reveal parenchymatous degeneration. The respiratory tract is commonly the seat of catarrhal inflammation.

In rare instances general infection occurs without intestinal lesions (*typhoid bacteremia* or *septicemia*).

Symptoms.—The *period of incubation* is from one to three weeks. In this period the patient's health may be unaffected, but as a rule, there are certain *prodromal symptoms*, such as malaise, chilliness, headache and backache, nose-bleed, anorexia, diarrhea, and cough.

The Attack.—Fever.—The temperature rises gradually, reaching its maximum (104°–105° F.) by the end of the first week; it remains at this elevation for another period of from ten days to two weeks, when a gradual defervescence begins and occupies a third period usually lasting about a week. Throughout its course the fever is characterized by marked daily remissions, the evening temperature being from one to three degrees higher than the morning temperature.

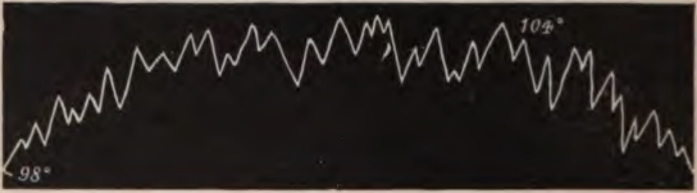

FIG. 12.—Temperature-curve in typhoid fever.

In some cases, especially in the young, the temperature rises quite abruptly. Slight diurnal remissions usually indicate a protracted case. As defervescence advances the temperature becomes more irregular; the remissions are more decided, and occasionally the higher temperature is recorded in the morning. An abrupt fall of several degrees should suggest intestinal hemorrhage or perforation.

Respiratory Symptoms.—These include hurried breathing, cough with tenacious sputum, and numerous bronchial râles.

Circulatory Symptoms.—As the disease progresses the pulse becomes frequent (90–130), weak, and dicrotic. In the early stages the rate is usually less than would be expected from the fever. At the height of the disease the systolic pressure averages about 110 mm. The heart-sounds become feeble. The first may be especially weak, and resemble the second.

The Face.—The expression is dull and heavy, the cheeks are somewhat flushed, the conjunctivæ are clear, and the pupils often slightly dilated.

The tongue is tremulous; at first it is red at the tip and edges, and covered posteriorly with a whitish fur. In severe cases the tongue becomes dry, brown, and fissured, and *sordes* collect on the teeth.

The Stomach.—Gastric symptoms are not common, but obstinate vomiting sometimes develops and becomes a serious complication.

Intestinal Symptoms.—The abdomen is usually distended. Tenderness is frequently noted on palpation; it may be general or confined to the right iliac fossa. Gurgling may also be detected in the latter region, but it has little significance. Diarrhea is usually present, though it is not a constant symptom. The discharges vary in number from three to six or more a day; they are thin, offensive, and of a yellowish color (likened to pea-soup); on standing, a turbid liquid rises to the top and a granular sediment falls to the bottom. Intestinal hemorrhage occurs in from 5 to 7 per cent. of the cases.

The Eruption.—This appears from the seventh to the ninth day, and is most abundant on the abdomen, though it is not infrequently observed on the chest and back. It is composed of small, slightly elevated, rose-colored spots that disappear on pressure. It comes out in successive crops over several days. It may be absent, particularly in the aged and very young. Rarely, in malignant cases, the eruption is petechial. Sudamina and miliaria are common. Herpes is rare.

Splenic enlargement is rarely absent. Rupture has occurred in a few instances.

Nervous Symptoms.—In mild cases apathy, headache, and slight deafness may be the only nervous symptoms. In severe cases there may be muttering delirium, stupor, twitching of the tendons (subsultus tendinum), picking at the bed-clothes or imaginary objects (carphologia), and ultimately coma.

The Blood.—The number of leukocytes is diminished (5000–1000). In the later stages the hemoglobin and the number of red cells are also more or less reduced. The blood-serum agglutinates the typhoid bacillus (*Widal reaction*). The reaction may be obtained at the end of the first week, and is to be considered positive if there is general clumping of the bacilli with a dilution of 1 to 50 in one hour. It may persist for months or years after recovery. The typhoid bacillus may often be grown from the blood before the Widal reaction is given.

The urine is usually scanty and slightly albuminous. In many cases (25 per cent) it contains typhoid bacilli. Retention is common.

Convalescence is marked by anemia, falling of the hair, desquamation of the cuticle, and sometimes by mental enfeeblement.

Varieties.—*Mild Typhoid.*—There is moderate fever with marked remissions; the diarrhea is slight; nervous symptoms are often absent; the rash is usually present, and may be abundant.

Abortive Typhoid.—There is an abrupt onset with severe symptoms, but convalescence follows in from ten days to two weeks.

Walking Typhoid.—The symptoms are mild, and often disregarded by the patient, who refuses to go to bed; but grave symptoms may develop suddenly, and death from perforation is not uncommon.

Typhoid in Children.—The rash is often absent; the temperature rises abruptly; cerebral symptoms are frequently marked; hemorrhage is comparatively rare; the course is usually mild.

Complications.—Any symptom aggravated constitutes a complication; thus high fever, excessive diarrhea, and tympanites may be troublesome complications.

Hemorrhage.—This usually occurs during the third week, and is indicated by a sudden fall of temperature, followed by dark-red or tarry stools. After large hemorrhages there may be symptoms of collapse.

Perforation.—This occurs in 2 to 3 per cent. of all cases and in from 10 to 20 per cent. of the fatal cases. The suggestive symptoms are localized pain and tenderness, fall of temperature, leukocytosis, a pinched expression, vomiting, marked tympanites, disappearance of the liver dulness, rigidity of abdominal muscles, vesical irritation, and signs of peritonitis.

Laryngitis, lobar pneumonia, hypostatic congestion of the lungs are common complications.

Among the less frequent complications or sequelæ may be mentioned: cholecystitis (not rarely leading to cholelithiasis), phlebitis, peripheral neuritis, pyelitis, nephritis, otitis media, furunculosis, parotitis, periostitis, spondylitis (typhoid spine), and posttyphoidal insanity (rarely permanent).

With the exception of myocarditis, cardiac complications are rare.

Relapse and Recrudescence.—*Relapses* are common (about 10 per cent. of all cases); they repeat the symptoms of the original attack, but are generally milder and of shorter duration, and seldom prove fatal.

Recrudescence.—This is a sudden temporary elevation of temperature occurring during convalescence, and is not associated with a return of the other symptoms. It is usually due to constipation, excitement, or irritating food.

Diagnosis.—*Acute miliary tuberculosis* often closely resembles typhoid fever. In tuberculosis the temperature is usually more irregular; the abdominal symptoms are less marked; pulmonary symptoms, especially dyspnea, are more marked; the rash is absent; the Widal reaction is absent; tubercles may occasionally be detected on the retina; and symptoms of basilar meningitis may be present, such as inequality of the pupils, ptosis, and strabismus.

Malignant Endocarditis.—The following features would suggest endocarditis: The history of a primary disease which might induce endocarditis; irregular fever; intercurrent rigors; marked leukocytosis; precordial pain and endocardial murmurs; embolic lesions; and the absence of a rose-colored rash, of the Widal reaction, and of marked abdominal symptoms.

Enteritis.—The absence of high fever, of eruption, of splenic enlargement, of epistaxis, and of bronchial catarrh will serve to distinguish enteritis from typhoid fever.

Meningitis.—The abrupt onset, the early development of cerebral symptoms, the irregular fever, the leukocytosis, and the absence of the characteristic rash, of abdominal symptoms, and the Widal reaction will indicate meningitis.

Prognosis.—The prognosis is always doubtful. The mortality varies in different outbreaks of the disease, but the average is about 8 per cent.

A pulse-rate persistently above 120, excessive diarrhea, marked tympanites, severe nervous symptoms, hypostatic congestion of the lungs, and intestinal hemorrhage are unfavorable features.

Prophylactic Measures.—These comprise the obtaining of a pure water supply, the proper disposal of excreta, precautions against food (especially milk) contamination, protection against flies, the isolation of the sick, the thorough disinfection of the patient's discharges (feces and urine), and all articles (bed-linen, bed-pans, urinals, etc.) likely to be soiled by the patient, and constant care on the part of those who handle the sick to protect themselves and others against direct infection. During the prevalence of epidemics all drinking water and milk should be boiled and no vegetables or shellfish should be eaten raw unless their source is known to be above suspicion. Typhoid vaccination usually affords protection for two or three years, and is to be strongly recommended in the case of soldiers, nurses, orderlies and all others who are in special danger of contracting the disease. The vaccine is given hypodermically once a week for three weeks, the first dose consisting of 500 million killed bacteria, the second, 1000 million, and the third, 2000 million.

Treatment.—As soon as the nature of the disease is recognized the patient should be confined to bed. The room should be large and airy, and provided with efficient means of securing thorough ventilation. The temperature of the room should be maintained between 65° and 70° F. The bed-pan must be used from the beginning until convalescence is well advanced. The stools and urine should be rendered innocuous before being thrown out. This may be done by treating the evacuation with twice its volume of a 1 per cent. solution of chlorinated lime or a 5 per cent. solution of carbolic acid, and allowing it to stand in a covered vessel for two hours before emptying it into the closet. Soiled clothing should be thoroughly boiled.

The diet should be liquid or semisolid, unirritating, and easily digestible. Milk alone (6 ounces every four hours) does not supply the required number of calories (2500–3000), but, as a rule, it should form a large part of the diet. It may be given diluted with lime water, or as buttermilk, malted milk, koumiss, junket, or ice cream. Among other suitable foods may be mentioned raw or soft boiled eggs, chicken jelly, milk toast, strained oat meal gruel, potato puree, tea, coffee, cocoa, fruit juices, wine jelly, and custard. Beef tea and broths may be harmful. In the event of digestive disturbances the diet should be restricted for a time to whey or albumin-water. Water, plain or flavored (lemonade, soda-water, etc.), should be given in large amounts between the feedings. When the first sound of the heart becomes weak and the pulse dicrotic, alcohol is usually indicated. From 4 to 8 ounces of whisky or brandy may be given in the twenty-four hours, the amount being determined by the general effect.

The cold bath or the cold pack affords the best means of controlling fever and preventing the development of severe nervous symptoms. It may be employed every three or four hours when the temperature is 102.5° F. or over. Hemorrhage, signs of perforation, menstruation, and great prostration are contraindications.

Heart-failure.—Cold bathing and the timely use of alcohol do much to guard against heart-failure. When the tendency to cardiac failure is pronounced, strychnin may be given in doses of from 1/40 to 1/20 grain every three or four hours. In severe cases the drug should be given hypodermically. Digitalis may also be tried, but in the presence of fever it often proves ineffectual. Salt solution (500 c.c. to 750 c.c. once or twice a day, by the bowel or subcutaneously) is often of service. If collapse is threatened, ether, alcohol, or, better still, camphor (1 to 2 grains in sterile olive oil) may be given subcutaneously every two or three hours.

Diarrhea.—It is advisable to substitute albumin-water for all other food and to give bismuth subnitrate (20 gr.) with a mild antiseptic and codein by the mouth. If the diarrhea be troublesome, silver nitrate may be given by the mouth in combination with opium:

℞.	Codeinæ sulphatis	gr. ii–iv
	Beta-naphtholis	gr. xl
	Bismuthi subnitratis	ʒss.—M.

Fiant chartulæ No xii.
SIG.—One every three or four hours.

℞.	Argenti nitratis	gr. iij
	Pulveris opii.	gr. vj.—M.

Fiant pilulæ xij.
SIG.—One pill every three or four hours.

In very obstinate cases copper sulphate with opium in pill sometimes proves efficacious.

Constipation.—This may be relieved by enemas of soap and water or by the administration of fractional doses of calomel.

Tympanites.—This may often be relieved by reducing the diet to albumin-water, applying turpentine stupes, giving by the mouth small doses of creosote, turpentine, or betanaphthol, and using enemas containing asafetida or turpentine. Enteroclysis is sometimes useful. If extreme, a soft rectal tube may be introduced into the bowel. Physostigmin sulphate (1/60 grain twice a day) is of service.

Hemorrhage.—Absolute rest is imperative. Cold bathing should be suspended. It is advisable to elevate the foot of the bed. An ice-bag may be applied with advantage to the right iliac region, and ice may be given to suck. The best drug is morphin (1/6 to 1/4 grain) hypodermically. Ergot is useless. In cases of recurrent hemorrhage calcium lactate (10 grains thrice daily) and gelatin may be given by the mouth.

Insomnia.—Opium is usually the best somnifacient. In some cases, however, sodium bromid, veronal, or chloralamid acts better.

Delirium is best managed by hydrotherapy. Low, muttering delirium usually calls for alcoholic stimulation. An ice-cap is useful. Salt solution by the bowel, or in serious cases subcutaneously, is often serviceable. Camphor or musk may be tried: the former is best given hypodermically, the latter, by the bowel. In active or violent delirium no drug is so generally useful as morphin.

Retention of Urine and Bacilluria.—Retention of urine, a common difficulty, is sometimes overcome by the use of hot applications or an enema of hot water. In many cases, however, it is necessary to use the catheter. For the bacilluria, hexamethylenamin (5 to 10 grains thrice daily) is the best remedy.

Perforation.—Recovery from peritonitis is so exceedingly rare under medicinal treatment that operative interference is

Definition.—An acute i any characteristic anatom toxemia, a maculopapular chial, and high fever lasti by crisis.

Etiology.—Typhus fever one of the most dreaded epidemic. It is more or le and Ireland, Russia, south small outbreaks occur from cities. Overcrowding and factors. Until a few years be air-borne, but the resea Wilder, and others have den the chief agent (probably th disease. One attack usually

Pathology.—There are n other acute infections, the the parenchymatous organs changes. The blood is dark

Symptoms.—The *period of* two weeks. The disease beg pains, marked prostration, reaches its contracted ("ferret eyes"). As the disease progresses delirium, sometimes maniacal, usually develops, and in grave cases other nervous derangements, such as subsultus tendinum, carphologia, and coma vigil (coma with open staring eyes) supervene. The *rash* appears on the fourth or fifth day. It consists of a single crop of slightly elevated dull-red macules, which frequently become petechial within forty-eight hours. Between the macules there is in many cases a dusky-red mottling ("mulberry rash"). The urine is scanty, high-colored, and slightly albuminous. Leukocytosis is usually present. In the type studied by Brill nervous symptoms, with the exception of severe headache, are usually absent, and the eruption is rarely petechial.

Complications.—These are not very common, but bronchopneumonia, hypostatic congestion of the lungs, phlebitis, nephritis, and localized gangrene may occur.

Diagnosis.—*Cerebrospinal Meningitis.*—In this affection the pain in the back is greater; the temperature-curve is much more irregular; there is a marked tendency to opisthotonos and facial palsies; vomiting is much more common, and the eruption, though it may resemble that of typhus, is inconstant and without a special time for appearing.

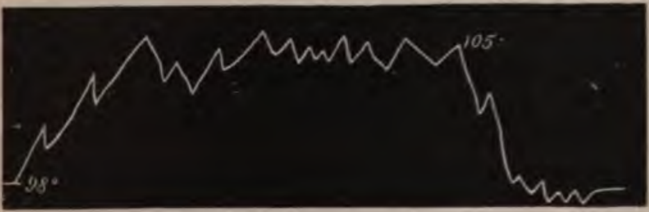

FIG. 13.—Temperature-chart of typhus.

Typhoid Fever.—The resemblance is in the nervous phenomena only. In typhoid fever the fever rises gradually and subsides by lysis; the eruption appears later, remains rose-red, and does not become petechial; the face is not dusky; the eyes are not injected; the blood yields the Widal reaction, and there are marked abdominal symptoms.

Prognosis.—The mortality varies in different epidemics. It may exceed 20 per cent. Advanced years and alcoholism render the prognosis very grave.

Treatment.—Destruction of body-lice is an important prophylactic measure. The patient should be isolated, his clothing disinfected, and his body sponged with a solution (1 to 2000) of corrosive sublimate; and the apartment, after his removal, should be thoroughly fumigated with burning sulphur. The general treatment of the disease does not differ from that of typhoid fever.

RELAPSING FEVER

(Spirillum Fever; Famine Fever)

Definition.—An acute infectious disease due to the Spirochæta of Obermeier and other species of spirochetes, and characterized by recurring paroxysms of high fever lasting for from five to seven days.

Etiology.—The disease prevails at times in Europe, Africa (African tick fever), and India. The last American epidemic was in 1869. It attacks especially young adults, and unsanitary conditions seem to favor its spread. The infection is apparently transmitted by the bed-bug, the body-louse, and certain species of ticks.

Pathology.—There are no characteristic lesions. The liver and spleen are enlarged, and the latter is frequently the seat of infarctions. The parenchymatous organs show cloudy swelling. There may be hyperplasia of the bone-marrow.

Symptoms.—The *period of incubation* is from five to ten days. The disease begins abruptly with a chill, followed by fever, which reaches its maximum (105°–106° F.) in twenty-four hours, and remains high for from five to seven days, when it falls by crisis. After an intermission of five or six days it again rises rapidly and remains high for a similar period. Convalescence usually begins at the end of the second paroxysm, but it may not begin until after the third or fourth. Other noteworthy symptoms are intense pains in the head, back, and joints, and the presence of the spirochæta in the peripheral blood during the febrile stage. Gastric irritability and jaundice are also common. Not infrequently there is an ecchymotic rash.

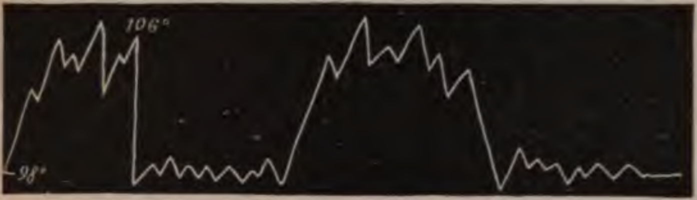

FIG. 14.—Temperature-curve in relapsing fever.

Complications.—These are uncommon, but there may be nephritis, pneumonia, ophthalmia, or hemorrhages from the kidneys, stomach, or intestine.

Diagnosis.—The characteristic febrile paroxysms with the long intermissions and the presence of the spirochæta in the blood are the distinctive features.

Prognosis.—In the absence of complications the prognosis is good. The mortality in different epidemics varies from 4 to 15 per cent.

Treatment.—The prophylactic measures do not differ from those employed against typhus fever (see p. 334). The treatment is purely symptomatic. Absolute rest, good nursing, and proper diet will do much to avert complications. Vomiting may be controlled by carbonated water, wine of ipecac (1 minim), diluted hydrocyanic acid (1 or 2 drops), or cocain (1/8 grain); the fever by cold sponging; and the severe pains by morphin or phenacetin.

CEREBROSPINAL FEVER

(Epidemic Cerebrospinal Meningitis; Spotted Fever)

Definition.—A specific infectious disease due to the Diplococcus intracellularis meningitidis, and characterized by inflammation of the cerebral and spinal meninges.

Etiology.—The disease may be sporadic or epidemic. Overcrowding and other unsanitary conditions predispose to epidemics. Outbreaks are most common in the winter and spring. The young are more susceptible than the aged. The disease is mildly contagious, and meningococcic "carriers" probably play an important part in its dissemination. Infection is believed to occur through the nose.

The Exciting Cause.—The Diplococcus intracellularis meningitidis of Weichselbaum is the specific cause of the disease. This organism appears in the polymorphonuclear leukocytes of the exudate, and may be gotten from the nasal secretion and the fluid obtained by spinal puncture.

An infection almost identical with cerebrospinal fever may be caused by other organisms, especially the Diplococcus pneumoniæ.

Pathology.—In most cases the membranes of the brain and cord are deeply congested and opaque. Serofibrinous or purulent exudation is found both at the base and on the convexity of the brain, especially in the fissures and along the blood-vessels. The spinal meninges present similar changes, the posterior surface of the cord being particularly involved. The cranial nerves are usually involved and in many cases the ventricles are distended with turbid fluid. The organs exhibit the changes commonly found in acute infections. In rapidly fatal cases the lesions may be very slight.

Symptoms.—**Common Form.**—The disease usually begins abruptly with a chill, followed by vomiting, excruciating pain in the head, back, and limbs, and fever. The muscles of the neck and back become rigid and contracted, so that the head is bent backward and the back is straightened; in severe cases the body may be arched in a state of opisthotonos. Kernig's sign is an almost constant phenomenon.[1] The temperature is irregular in its course and indefinite in its duration. Ordinarily it ranges between 101° and 103° F., but in some cases it remains nearly normal, and in other cases it is very high. It may become normal within a fortnight or it may persist for months. The pulse is also variable. It is usually accelerated, but it may be very slow (60–50). In advanced cases the respiration is sometimes of the Cheyne-Stokes type. The abdomen is often retracted and the bowels are usually constipated. The urine may be slightly albuminous. Leukocytosis (20,000 to 40,000) is the rule. Delirium is rarely absent and in severe cases it is followed by stupor and coma. Convulsions often occur. In the majority of cases emaciation is rapid and progressive.

Involvement of the Cranial and Spinal Nerves.—Evidence of involvement of the cranial nerves may be found in nystagmus, strabismus, ptosis, inequality of the pupils, and partial deafness or blindness. Involvement of the spinal nerves is shown in extreme cutaneous hyperesthesia, tonic spasms of the flexor muscles, and later, at times, in paralysis of the extremities.

The Eruption.—The eruption is neither constant nor characteristic. In many cases a blotchy purpuric eruption appears over the entire body. Herpes facialis is also frequently observed. In other cases an urticarial or a roseolar rash appears.

Lumbar Puncture.—Fluid obtained by lumbar puncture is usually turbid and contains the diplococcus and a preponderance of polynuclear cells.

Fulminant Form.—There is an abrupt onset, with a chill, followed by vomiting, headache, moderate fever, convulsions, a petechial or purpuric rash, and death within twenty-four or thirty-six hours from collapse.

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[1] This consists in an inability to straighten the leg completely when the patient is in the recumbent posture and the thigh is flexed at a right angle with the pelvis.

Abortive Form.—The disease begins abruptly with grave symptoms, but terminates in a few days in recovery.

Intermittent Form.—In this form intermissions or marked remissions occur daily or every other day in the fever and other symptoms.

Chronic Form.—In some cases the acute symptoms subside, but the patient remains in a stuporous state for many months and ultimately presents extreme emaciation.

Diagnosis.—*Typhoid Fever.*—The gradual onset, the regular fever, the diarrhea and tympanites, the Widal reaction, and the absence of rigidity, of intense pain in the back and limbs, of facial palsies, of leukocytosis, of Kernig's sign, and of herpes will serve to distinguish typhoid from cerebrospinal fever.

Typhus Fever.—The regular fever, the absence of intense pain in the back and limbs, of facial palsies, of Kernig's sign, and of muscular rigidity will distinguish typhus from cerebrospinal fever.

Acute articular rheumatism may resemble cerebrospinal meningitis, but the early involvement of the joints, the acid sweats, and the absence of rigidity, of eruption, and of facial palsies, will distinguish it from cerebrospinal meningitis.

Tuberculous Meningitis.—In this disease the onset is less abrupt; there is less tendency to opisthotonos; herpes is rare; petechiæ are absent. Lumbar puncture affords a reliable means of diagnosis and a primary focus of tuberculosis can usually be detected elsewhere in the body.

Prognosis.—The mortality has varied in different epidemics from 25 to 75 per cent. Under serum treatment it probably does not exceed 25 per cent. The cases without severe cerebral symptoms are the most favorable.

Complications and Sequelæ.—These include defective vision from inflammation of the cornea or retina or from atrophy of the optic nerve; defective hearing from inflammation of the auditory nerve or from suppurative inflammation of the internal or middle ear; pneumonia; arthritis; aphasia;

peripheral palsies; imbecility; chronic hydrocephalus; and persistent headache from chronic meningitis.

Treatment.—The patient should be isolated in a quiet, darkened, and well-ventilated room. The diet should be liquid and supporting. In some cases, in order to secure the ingestion of enough nourishment, it may be necessary to resort to nutrient enemas or forced feeding by means of a stomach-tube. Cardiac failure must be combated by stimulants, of which the best is whisky or brandy.

In sthenic cases the withdrawal of several ounces of blood by wet-cups applied along the cervical vertebræ may prove useful. Cold applied to the head and along the spine affords considerable relief. Blisters to the nape of the neck are of doubtful value, at least during the irritative stage. Morphin hypodermically is the best drug for the relief of pain, restlessness, spasms, and insomnia. In mild cases bromids may suffice.

Repeated lumbar punctures have been found useful in relieving excruciating headache, delirium, somnolence, and coma. The serum of Flexner and Jobling has given very encouraging results. From 30 to 40 c.c. are injected in the spinal canal after an equal amount of cerebrospinal fluid has been withdrawn, and it is advised that the treatment be repeated once a day for three or four days. Warm baths (105°-110° F.) twice a day for ten minutes have been highly recommended.

Tonics—iron, strychnin, and cod-liver oil—are generally indicated during convalescence. Local palsies will require massage and electricity.

MALARIAL FEVER

(Chills and Fever; Fever and Ague; Paludism

Definition.—An infectious disease, excited by a protozoan parasite, first described by Laveran (Plasmodium malariæ), and characterized by splenic enlargement, fever with periodic intermissions or remissions, chills, and anemia.

Etiology.—Man becomes infected through the bite of certain mosquitos, namely, those belonging to the genus Anopheles, which serve as hosts for the parasite. The usual source from which the mosquito derives the parasite is man. The conditions predisposing to infection are those which are favorable to anophelinæ, namely, high temperature, humidity, and natural collections of water undisturbed by winds or currents.

Persons living in high-lying localities are less liable to infection than those living in low lands, because the mosquito does not rise high above the ground. Malaria is more likely to be contracted at night than during the day, because most species of anopheles are nocturnal in their habits. Males being more exposed to infection are more often attacked than females.

Parasitology.—At least three distinct species of *Plasmodium* are recognized in man: *Plasmodium vivax*, causing tertian fever; *Plasmodium malariæ*, causing quartan fever; and *Plasmodium falciparum*, causing estivo-autumnal fever or tropical malaria.

The infected mosquito introduces the spores (*sporozoites*) into the system of man. In the red blood-cell the hyaline spore develops into an ameboid nucleated form, using the hemoglobin of the corpuscle as food, and finally leaving only a residue of blood-pigment (melanin). In twenty-four, forty-eight, or seventy-two hours, according to the species, the mature parasite divides into a number of daughter cells, forming a rosette around a central clump of pigment. The red blood-corpuscle then disintegrates liberating the daughter cells and also a toxin, which is the cause of the malarial paroxysm. The free daughter cells (*sporozoites*) enter other red blood-cells and repeat the sporulating cycle. Although male and female elements exist among the sporozoites, segmentation in man is wholly asexual. Sporozoites are produced sexually only in the mosquito.

In the stomach of the mosquito the male and female elements unite to form a vermicule, a mobile form, which enters the stomach-wall, where it develops into a sac (zygote) containing pear in the blood. These bodies are characteristic of estivo-autumnal infection and represent sexually differentiated forms, or gametocytes, which breed in the stomach of the mosquito. There appear to be at least two varieties of the estivo-autumnal parasite, one completing its life cycle in twenty-four hours, the other, in forty-eight hours.

Pathologic Effects.—The destruction of the red cells by the parasites is followed by anemia, melanemia, and pigmentation of the organs. The spleen becomes greatly enlarged from congestion. In chronic cases (malarial cachexia) it becomes hard and tough from hyperplasia of the fibrous tissue. Extreme disintegration of the blood may occasion thrombosis of small vessels and also hemoglobinuria.

Clinical Varieties of Malaria.—The following clinical forms are recognized: (1) Intermittent malarial fever; (2) estivo-autumnal fever; (3) pernicious malarial fever; (4) malarial cachexia (chronic malaria); (5) latent and masked malaria.

INTERMITTENT MALARIAL FEVER

Intermittent malarial fever is excited by tertian or quartan parasites. It is characterized by paroxysms of fever occurring at definite periods, each paroxysm consisting of a cold, a hot, and a sweating stage.

Cold Stage.—This stage is characterized by lassitude, aching in the limbs, and great chilliness. The features are pinched; the lips are blue; and the surface is cold and rough (cutis anserina). The rectal temperature, however, is high (105°–106° F.). Vomiting may occur. The chill may last from a few minutes to an hour or more.

Hot Stage.—The surface temperature gradually rises; the skin becomes hot; the face flushed; the eyes injected; and the pulse full and rapid. The temperature in the axilla may reach 106° or 107° F. The patient complains of severe pain in the head, back, and limbs, and of intense thirst. The urine is scanty and dark colored. This stage usually lasts from one to five hours.

type). If the localization is gastro-intestinal, there may be vomiting and purging of serous material, cramps, suppression of urine, coldness of the surface, profuse sweating, and fatal collapse (*algid type*). In other cases, in consequence of a sudden and intense hemolysis, the paroxysms are associated with jaundice, bilious vomiting, and hemoglobinuria. Bleeding into the subcutaneous tissues and from the mucous membranes may also occur (*hemorrhagic type*).

The infection known as *hemoglobinuric* or *blackwater fever*, prevailing in Africa, Italy, Central America, and our own Southern States, is undoubtedly related in some way to malaria, but whether the plasmodium is the sole cause of the syndrome or not is a mooted question.

Malarial Cachexia.—Malarial cachexia may be a sequel of repeated attacks of intermittent or estivo-autumnal fever, or it may develop insidiously as a primary condition.

There is pronounced anemia with its attending phenomena. Pigment granules are found in some of the leukocytes and in the plasma. The parasites are at times absent from the blood. The complexion is sallow or muddy. The temperature is usually subnormal, but there may be occasional slight attacks of fever. The spleen is greatly enlarged. Weakness and emaciation are marked. Indigestion, flatulency, and constipation are common symptoms. Periodic headache, neuralgia, and hematuria are sometimes observed.

Latent and Masked Malaria.—In latent malaria the parasites can be demonstrated in the blood, but no symptoms are present. In masked malaria, the parasites are present in the blood and symptoms are present, but the latter are atypic, often taking the form of headache, neuralgia, diarrhea, or dysentery.

Diagnosis of Malarial Infections.—Estivo-autumnal fever sometimes resembles *typhoid fever*. The latter may be recognized by the marked abdominal symptoms, typical rash, Widal reaction, and the absence of the malarial parasite.

Yellow Fever.—The comparatively slow pulse, early albuvalue, but, being distinctly less efficacious than quinin, they should be employed only when the latter is not well borne.

In ordinary *intermittent fever* the quinin should be given in daily doses of from 15 to 20 grains, so divided that the last dose is taken about two hours before the expected chill.

The remedy should be continued in full doses until the paroxysms fail to appear, and then gradually withdrawn over a period of several weeks. The administration of a laxative dose of calomel as a preliminary measure increases the efficacy of the quinin, probably by facilitating its absorption.

For adults quinin is best prescribed in capsules, cachets, or freshly made pills. For children it may be given suspended in syrup of chocolate or elixir of licorice. During convalescence iron and arsenic may be advantageously given with the quinin, as in the following formula:

℞.	Ferri pyrophosphatis	gr. xxx
	Arseni trioxidi	gr. ss
	Quininæ sulphatis	gr. xl
	Pulveris capsici.	gr. x.—M.

Pone in capsulas, No xx.
SIG.—One thrice daily after meals.

In *estivo-autumnal fever* larger doses of quinin (30 to 40 grains a day) are usually required. In *pernicious malarial fever* the patient should be cinchonized as quickly as possible by injecting at once into the tissues of the thigh or buttock about 30–40 grains of a soluble salt of quinin like the dihydrochlorate.

Symptomatic Treatment.—During the cold stage of the paroxysms the patient should be well covered with warm blankets and given hot drinks. Opium in the form of paregoric is sometimes useful in mitigating discomfort. It may be combined with a few minims of aromatic spirits of ammonia, chloroform, or Hoffmann's anodyne. In the hot stage much relief is afforded by frequently sponging the body with cool water, giving cold drinks, and administering, if the sy
oms are very severe, a sr

reveal ganurlar degeneration. The lymph-nodes in all situations are enlarged. The kidneys frequently show the lesions of hemorrhagic nephritis, the glomeruli being especially involved. The rash is rarely detected after death.

Varieties.—(1) Simple; (2) anginoid; (3) malignant.

Symptoms.—The *period of incubation* is from three to seven days, the *invasion* is sudden and marked by sore throat, vomiting and a rapid rise of temperature. In infants there is sometimes a convulsion and in adults there is often a chill.

Throat Symptoms.—These consist in pain and difficulty in swallowing; fullness and tenderness beneath the jaw; and enlargement of the lymphatic glands. The tongue is at first heavily coated, and red at the tip and edges; in a few days the coating almost entirely disappears, and the papillæ become bright red and swollen. This appearance has given rise to the term "strawberry tongue." The pillars, tonsils, uvula, and pharyngeal vault are deeply injected and may reveal a punctiform efflorescence before the rash develops on the skin. In severe cases the tonsils may be the seat of follicular inflammation, or may be covered with false membrane.

Eruption.—A scarlet-red punctiform rash appears at the end of the first or at the beginning of the second day, on the neck and chest, and rapidly spreads to other parts of the body. Though the forehead and cheeks are often intensely flushed, the region around the mouth usually appears pale. The rash disappears on pressure, a white line remaining for a second or two when the finger-nail is drawn through it. It may be uniform or it may occur in discrete patches surrounded by healthy skin. It lasts for from five to seven days, and is followed by flaky desquamation, which is usually completed in five or six weeks.

In some cases the rash is pale and scarcely visible, and in others it is slightly papular or vesicular (scarlatina miliaris).

Febrile Symptoms.—The fever rises rapidly, reaching its maximum (104°–105° F.) in from twenty-four to forty-eight hours, remain [illegible] ee or four days, and

Diagnosis.—*Acute tonsillitis* may resemble scarlet fever, especially if the former is associated with an erythematous rash; but in tonsillitis there is no history of contagion; the pulse is proportionate to the fever; the rash, if present, is not punctiform; the tongue has not the strawberry appearance; and there is little tendency to nephritis.

Diphtheria.—The onset is less abrupt; there is more prostration; false membrane, containing the Klebs-Löffler bacillus, is always present; a cutaneous rash is usually absent; and the tongue does not present a strawberry appearance.

Measles.—The sore throat is less marked; catarrhal symptoms are present; the rash appears later, is papular, and forms crescentic-shaped patches; the fever shows a decided remission on the second or third day; Koplik's spots are to be seen in the mouth, and the pulse is proportionate to the fever.

Rubella.—This may be difficult to distinguish from scarlatina, but the fever is not so high nor the pulse so rapid; the postcervical glands are more swollen; the strawberry tongue is absent; there is no tendency to nephritis, and the rash is not punctiform.

Acute Exfoliative Dermatitis.—In this disease there is often a history of previous attacks, the intensity of the eruption is out of all proportion to the degree of constitutional disturbance, sore throat and strawberry tongue are absent, albuminuria is uncommon, desquamation begins very early and the nails and hair often exfoliate.

Accidental Rashes.—Certain drugs, like belladonna, quinin, and copaiba, and certain foods, like crabs and oysters, may produce a rash like that of scarlet fever, but it is not punctiform, and is not associated with high fever, sore throat, and rapid pulse.

Prognosis.—This should always be guarded. The mortality varies in different epidemics from 5 to 30 per cent.

Treatment.—The patient should be isolated for from six to eight weeks. All articles used in the sick-room should be thoroughly disinfected before being removed. To allay the tympanic membrane bulges, indicating the presence of pent-up pus, the latter should be evacuated by puncture.

Should severe *nephritis* develop, dry cupping over the loins, followed by warm fomentations, will often prove of value. Aperients, especially salines, are indicated. Warm baths, hot packs, vapor-baths, or pilocarpin (1/16 to 1/10 grain) should be used to promote diaphoresis. If the urine is scanty, unirritating diuretics, like potassium acetate or bitartrate and digitalis, are of service.

MEASLES

(Rubeola; Morbilli)

Definition.—An acute contagious disease, characterized by catarrh of the respiratory tract, moderate fever, and a red papular eruption, which appears on the fourth day, lasts four or five days, and is followed by bran-like desquamation.

Etiology.—Measles is highly contagious, and the poison may be transmitted through clothes and other fomites. The contagium is apparently associated with the nasal and bronchial secretion, but it has not been isolated. Measles is most commonly observed in children, but unprotected adults are very liable to be attacked. It is endemic in large cities and every few years it becomes epidemic. One attack usually confers immunity against subsequent attacks.

Pathology.—The lesions consist in catarrh of the entire respiratory tract. Gastro-intestinal catarrh is not uncommon. In fatal cases such complications as bronchopneumonia and pulmonary collapse are frequently observed.

Symptoms.—The *period of incubation* is from seven days to two weeks. The invasion is characterized by *catarrhal symptoms*—photophobia, redness of the eyes, increased lacrimation, sneezing, discharge from the nose, hoarseness, cough, and, in older children, expectoration.

The Fever.—The temperature rises rapidly to 103° or 104° F., but on the second day there is often a decided remission which continues until the fourth day, when the eruption appears; at this time it again rapidly runs up to or beyond its original height, where it remains for three or four days and then falls by rapid lysis or crisis.

The Eruption.—This appears about the third or fourth day on the face, and rapidly spreads over the entire body. It is composed of small, dark-red, velvety papules, which tend to form groups having crescentic borders. There is often much itching of the skin. In three or four days the eruption begins to fade, and a branny desquamation soon follows.

Minute bluish-white specks surrounded by red aerolæ may be seen in strong daylight on the mucous membrane of the cheeks and lips one or two days before the skin eruption appears (Koplik's sign).

Malignant or Hemorrhagic Measles.—This form occurs usually in asylums, prisons, etc., where the conditions are unsanitary. It is characterized by a petechial rash, hemorrhages from the mucous membranes, and profound prostration.

Complications and Sequelæ.—Bronchopneumonia and acute gastro-intestinal catarrh are the most common complications. Among the less frequent complications or sequelæ may be mentioned membranous or ulcerative laryngitis, inflammation of the middle ear, chronic conjunctivitis, pulmonary tuberculosis, cancrum oris, and neuritis.

Diagnosis.—*Rubella.*—The preeruptive stage is very short; fever and catarrh are slight; sore throat is marked. The rash appears on the first or second day as a diffuse red blush or as small, pale-red spots that do not form crescentic-shaped patches; desquamation is scarcely noticeable.

Scarlet Fever.—The onset is more sudden and is marked by vomiting; there is severe sore throat instead of a general catarrh; Koplik's sign is absent, but the tongue is characteristic; the rash appears on the first or second day, is a diffuse punctiform erythema; the pulse rate is out of proportion to the fever, and there is much greater tendency to nephritis.

Prognosis.—Recovery is the rule in ordinary uncomplicated cases. Complications are liable to occur, however, and render the prognosis grave.

Treatment.—The child should be quarantined for at least four weeks from the onset of the disease. The sick-room should be well ventilated and moderately darkened. At least two weeks should be spent in bed.

Milk, junket, fruit-juices, broths, eggs, and gruels are suitable forms of nourishment. Water should be proffered at frequent intervals.

Daily inunctions of the body with cold cream or cocoa-butter will serve to allay itching of the skin. If the conjunctivitis is marked, the eyes should be protected with dark glasses and frequently cleansed with a solution of boric acid (15 grains to the ounce). Hot baths and hot drinks are indicated if the rash is delayed.

High temperature is best controlled by cold sponging or cold packs. Diarrhea will call for bismuth subnitrate and antiseptics like salol or bismuth-beta-naphthol. If there is severe bronchial catarrh, expectorants with sedatives, like paregoric, are indicated. The following mixture will be found useful:

℞.	Potassi citratis	ʒij
	Tincturæ opii camphoratæ	fʒij–iv
	Glycerini	f℥j
	Aquæ	q. s. ad f℥iij.—M.

Sig.—A teaspoonful every two hours for a child of three years.

During convalescence tonics—iron, strychnin, and cod-liver oil—are indicated.

RUBELLA

(**Rötheln; German Measles; Epidemic Roseola**)

Definition.—An acute contagious disease resembling both scarlet fever and measles, but differing from these in its short course, slight fever, and freedom from sequelæ.

Etiology.—The disease is highly contagious and may be ınsmitted by direct contact or by fomites. It is most quently observed in children, but unprotected adults are t exempt. It is essentially epidemic. One attack usually otects from another, but not from measles or scarlet fever.

Symptoms.—The period of incubation is from five days to ree weeks. Prodromes are slight or altogether absent. ıe disease begins with drowsiness, slight fever, and sore roat. The eruption appears on the first or second day, d varies considerably in its character. In some cases the sh is composed of pinkish-red, scarcely elevated papules, iich are more or less discrete (*rubella morbilliforme*); in hers the rash is bright red and diffuse, like that of scarlet ver (*rubella scarlatiniforme*). It spreads rapidly, lasts two three days, and is succeeded by slight desquamation. part from the sore throat, the catarrhal symptoms are ght. A very constant feature is marked swelling of the stcervical glands.

Prognosis and Treatment.—Complications are rare, and the vast majority of cases the disease ends favorably within week. The treatment is that of measles.

SMALLPOX

Variola

Definition.—An acute contagious disease characterized a sudden onset, an eruption passing successively through e stages of macule, papule, vesicle, pustule, and crust, and gh fever, exhibiting a distinct remission on the third or urth day.

Etiology.—The infection is usually conveyed by direct posure, but it may be transmitted by a third person or by mites. Unless protected by vaccination or a previous tack, nearly everyone, from the child *in utero* to the aged, is sceptible to the contagion. The colored race seems to be pecially predisposed. Epidemics occur chiefly in winter.

A certain protozoön, *Cytorrhyctes variolæ*, originally described by Guarnieri and present in two forms—one cytoplasmic and the other intranuclear—is regarded by some authorities as the specific cause of the disease. Pyogenic cocci occur in the pocks as secondary invaders.

Pathology.—The development of the variolous vesicle is the result of a peculiar degeneration of the protoplasm of the epithelial cells of the rete mucosum. There is a reticular degeneration of the cytoplasm with a more advanced degeneration of the nucleus. Genuine pocks are frequently found in the mouth, esophagus, nose, and larynx. Even the trachea and bronchi may contain them. The spleen is engorged. Cloudy swelling is present in the parenchymatous organs, and the spleen, bone-marrow, and the lymph-nodes are the seat of a marked cellular (mononuclear) proliferation.

Varieties.—Discrete, confluent, purpuric, varioloid.

Symptoms.—*Discrete or Ordinary Smallpox.*—The period of incubation is from ten days to two weeks. The invasion is sudden and manifested by chill, vomiting, high fever, headache, and intense lumbar pain. The temperature rises quickly to 103° or 104° F., and continues high for about three days, or until the variolous eruption is well developed, when it falls several degrees; the remission, which is accompanied by a marked improvement in the symptoms, lasts until the eruption becomes pustular (seventh or eighth day), and then the fever returns. This secondary (suppurative) fever is maintained with marked fluctuations, until the beginning of

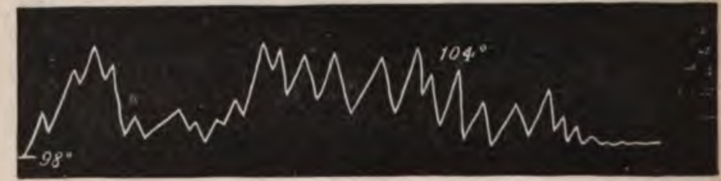

FIG. 16.—Temperature-curve in smallpox.

desiccation, when it gradually subsides, the temperature becoming normal about the end of the second week. The pulse is full and rapid (110–130); the respiration is quickened; the bowels are usually constipated; and the urine is often albuminous.

The Eruption.—In some cases a prodromal rash of a measly or scarlatiniform character is noticed on the first or second day. The true variolous eruption develops on the third or fourth day, appears first on the forehead and wrists, and spreads rapidly over the entire body. It begins as small red macules, which are quickly transformed into smooth hard papules. At the end of two days the papules become clear vesicles, centrally depressed (umbilicated), and divided by septa into several compartments, a single puncture failing to empty them. The vesicles gradually acquire a yellowish tinge and by the seventh or eighth day of the disease are converted into tense globular pustules, surrounded by red areolæ. Between the pustules the skin is edematous. In three or four days the pustules dry up or break, forming reddish-brown crusts, which emit a sickening odor and excite intense itching. In the course of two or three weeks the crusts or scabs separate leaving permanent pits or pock-marks.

Confluent Smallpox.—The papules are abundant and soon coalesce. The extremities are swollen and painful. The secondary fever is high and of long duration. True pocks nearly always develop in the air-passages and give rise to a copious fetid discharge from the nose and throat, to hoarseness, and to cough. Delirium, stupor, and subsultus are frequent symptoms. If the patient recovers, it is after a tedious convalescence, with great facial disfigurement, and often with defective vision or hearing.

Purpuric Smallpox.—In some cases the disease is ushered in with high fever, lumbar pains, and great prostration. Soon ecchymoses appear on the skin; bleeding from the mucous membranes follows; and death results before a true variolous rash appears. In other cases the disease advances like ordinary smallpox up to the pustular stage; then the pustules becomes effused with blood, and bleeding from the mucous membranes follows. This form is also very fatal.

Varioloid.—This is modified smallpox occurring in one who has been partially protected by previous vaccination. The symptoms are mild; the eruption resembles that of ordinary smallpox, but it is usually scanty and of short duration; secondary fever is absent.

Complications.—The most common complications are boils, ulcerative laryngitis, bronchopneumonia, and inflammations of the eye (conjunctivitis, ulcerative keratitis, iritis).

Diagnosis.—*Varicella.*—In this disease prodromes are generally absent and the constitutional symptoms are mild. The eruption appears on the first day; it comes out in successive crops; and, unlike that of smallpox, prefers the covered surfaces. It may be maculopapular at first, but it becomes vesicular within a few hours. The lesions are superficial; vary greatly in size; are usually unilocular, and are rarely umbilicated. Desiccation begins in two or three days, the vesicles becoming irregularly puckered at the periphery and presenting a depressed blackish crust in the center—a highly characteristic appearance. In doubtful cases in children the presence of a typical vaccinal cicatrix constitutes strong presumptive evidence against variola.

Syphilis.—The history of infection; the associated evidences of syphilis (mucous patches, alopecia, etc.); the gradual onset of the illness; the slight fever; the symmetric distribution of the eruption; its dark coppery color; its polymorphous character (papules, vesicles, and pustules associated in a limited area); and the absence of itching will indicate syphilis.

Prognosis.—This depends upon the virulence of the epidemic, the vaccinal condition of the patient, and the amount of eruption. In the discrete cases the prognosis is generally favorable; in the confluent, very grave; and in the purpuric almost hopeless. In the unvaccinated the mortality ranges between 20 and 60 per cent. Among those having one or two typical vaccine scars, the death-rate is very low—usually less than 7 per cent.

Treatment.—The preventive measures against smallpox include the complete isolation of the patient (preferably in a special hospital), the thorough disinfection of all objects that have been in contact with him, and, above all, the vaccination of every one who has been or who is likely to be exposed to the contagion. Absolute rest in bed, light bed-clothing, a well-ventilated room of a temperature of 65° F., an easily assimilable but sustaining diet, and the free use of cool drinks are requisites of treatment. The severe lumbar pains will require opium and the application of hot-water bags. Fever is best combated by hydrotherapy—cold sponging, cold packs, or cold baths. Antipyretic drugs should be used with caution.

Gastric irritability may be controlled by diluted hydrocyanic acid (2 minims), subnitrate of bismuth (10 grains), or cocain (⅛ grain). If nervous symptoms are not relieved by hydrotherapy, opium with bromids or chloral with bromids should be tried. Alcoholic stimulants are frequently demanded, especially in confluent cases.

An attempt should be made to keep the nasopharynx clean by means of antiseptic sprays or douches. The eyes should also be kept clean by frequent applications of a warm boric-acid solution (15 grains to the ounce).

Prevention of Pitting.—The room should be darkened and the exposed parts covered with cloths wrung out of a weak solution of carbolic acid (1 : 200) or of corrosive sublimate (1 : 5000 or 1 : 10,000). Unfortunately, when the lesions are deeply seated, there are no efficient means of preventing pitting.

In the stage of desiccation, warm baths followed by inunctions with cold cream or olive oil are useful in allaying itching and in hastening the removal of the crusts.

VACCINIA

(Vaccination; Cow-pox)

Definition.—A general disease with a local manifestation resembling the pock of variola, and acquired by inoculation with the virus of cow-pox. It is probably smallpox modified by passage through the cow.

History and Object.—The value of vaccination as a means of protection against smallpox was first made known to the world in a paper published by Edward Jenner in 1798.

Recent vaccination gives almost complete immunity against variola; the mortality of smallpox acquired after vaccination is almost inversely proportionate to the number of true vaccine scars.

Etiology.—Vaccinia is induced by inoculating the arm or leg with fresh virus obtained from the udder of a calf suffering from cow-pox (bovine virus). Formerly virus taken from a human vaccine vesicle was also employed (humanized virus), but on account of the risk of transmitting syphilis and other diseases, this source has been practically abandoned.

It has been shown that the addition of glycerin to vaccine lymph serves to preserve it and to free it from pathogenic bacteria.

Time of Performance.—The first vaccination should be performed, as a rule, about the fourth or fifth month, the second at the seventh year, and the third at puberty. Vaccination should always be repeated when smallpox is prevalent.

Performance of Vaccination.—The part selected should be thoroughly cleaned with soap and water, then with alcohol, and finally with pure water. A number of cross-scratches should next be made over an area about ⅛ of an inch in diameter, with a sterilized needle or special scarificator, deep enough to allow of a little oozing of pinkish serum. The virus should then be applied and well rubbed into the exposed lymph-spaces by additional scarification. A shield nay be worn for a few hours or until the wound has become erfectly dry; after that it should be discarded.

Symptoms.—About the third or fourth day after the peration a papule surrounded by a red areola forms at the seat of inoculation. By the fifth or sixth day the papule becomes a vesicle with a distinct central depression (umbilication). By the eighth day the vesicle is perfected and is then surrounded by a wide zone of inflammatory edema, which is the seat of intense itching. About the tenth day the areola begins to fade, and desiccation sets in with the gradual formation of a thick brown crust, which becomes detached in the fourth week. The scar is at first red, but in the course of several months it becomes paler than the surrounding skin. It has a punched-out appearance and is pitted.

The evolution of the pock is usually accompanied by malaise, slight fever, and enlargement of the axillary glands.

Complications.—These are uncommon. Abscess, erysipelas, or even tetanus may occur from secondary infection. Roseolar or urticarial eruptions are occasionally seen. Generalized vaccinia occurring spontaneously or resulting from autoinoculation is sometimes observed.

VARICELLA

(Chicken-pox)

Definition.—An acute contagious disease of short duration, characterized by slight fever and a discrete vesicular eruption, which disappears in two or three days by desiccation.

Etiology.—The disease occurs sporadically and epidemically. It is observed chiefly in children, but adults are not exempt. One attack usually protects from others. It bears no relation to smallpox.

Symptoms.—The *period of incubation* is from fourteen to sixteen days. The constitutional disturbance is usually slight. In most cases, however, there is slight fever (99°–101° F.) with chilliness and malaise.

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Diagnosis.—The different
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Prognosis and Treatment.
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DIPH

Definition.—An acute con
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branes, especially those of the

Etiology.—The disease is en
during the colder months of t
ally. The infection may be

children under ten years of age. One attack confers only temporary immunity.

The exciting cause is the Klebs-Löffler bacillus—the *Bacillus diphtheriæ*—which is found chiefly in the affected mucous membranes. The local lesions and the constitutional disturbances are caused by toxins, elaborated by the bacillus. The disease is essentially a local infection with a general toxemia. Secondary infection by pyogenic and other organisms is common.

Pathology.—The false membrane is usually found on the tonsils, pillars, and pharynx, but it may extend to the mouth, larynx, or nose. It has a grayish or yellowish appearance, is firmly attached to the underlying tissues, and when forcibly removed, leaves a raw and bleeding surface. Microscopically, it is composed of fibrin, necrotic epithelium, leukocytes and bacteria.

The lymphatic glands near the seat of infection are swollen. Focal necrosis, due to the action of the toxins, is found in the liver and other organs. The heart, kidneys, and liver are the seat of fatty and parenchymatous degeneration. Interstitial hemorrhages are frequently observed and are the result of hyaline degeneration of the capillary walls and thrombotic obstruction. Such lesions as congestion, edema, bronchopneumonia, and atelectasis are frequently encountered in the lungs.

Types.—Diphtheria may be divided according to the location of the exudate into: (1) Faucial; (2) laryngeal; and (3) nasal. According to the severity of the attack it may be divided into: (1) Mild; (2) grave; (3) malignant.

Symptoms.—*Faucial Diphtheria.*—The *period of incubation* is from two to seven days. The disease usually begins with chills, moderate fever, malaise, and sore throat. The fever, as a rule, is not very high (102°–104° F.), and its course is quite irregular. The pulse is rapid and feeble; the bowels are constipated; the urine is scanty and frequently albuminous; and the prostration and pallor are often out of all proportion stripped off, exposes a raw may spread to the nose or l

The course of the disea duration is from ten days t

In malignant cases there areas and of the lymph node gangrenous appearance and intense. Death occurs withi

Laryngeal Diphtheria (*M* ally secondary by extension sionally primary. It is reco croupy cough, progressive d The alæ of the nose pla prominent; the suprasternal of the chest is retracted. Sh times expectorated in the pulse is rapid and feeble, bu Death often results from suf possible even in the most un

Nasal Diphtheria.—This is it is characterized by a pr from the nose, epistaxis, an media, and suppuration of the lymph nodes. The most important sequel is paralysis, due to toxic neuritis. This occurs in about 20 per cent. of the non-fatal cases and usually appears during the second or third week of convalescence. The pharynx is the most common seat, the palsy being manifested by difficulty in swallowing and the return of liquid through the nose. The external muscles of the eye are often involved, the result being ptosis or strabismus. In some instances the heart is affected and if sudden death does not ensue the condition may be manifested by tachycardia or bradycardia. The muscles of the extremities may also be involved. The paralysis usually disappears in from a few weeks to several months.

Diagnosis.—*Scarlet Fever.*—This may be distinguished by the characteristic strawberry tongue, the very rapid pulse, the diffuse punctiform rash, and the absence of the diphtheria bacillus.

Follicular Tonsillitis.—The differential diagnosis between this disease and diphtheria has already been considered (see p. 33).

Prognosis.—The outlook depends upon the severity of the attack and the promptness with which treatment is instituted. The disease is very fatal during the first two years of life. The average mortality at the present time ranges between 15 and 20 per cent. The nasal and laryngeal forms are always grave. Death may be due to exhaustion from the toxemia, involvement of the larynx, bronchopneumonia, cardiac paralysis, or nephritis.

Treatment.—*Prophylaxis.*—As diphtheria is prone to attack unhealthy mucous membranes, nasopharyngeal catarrh in children should receive careful attention. Large tonsils and adenoid growths should be removed. Those who have been exposed to the contagion should receive immunizing doses of antitoxin (1000 units). Two weeks is probably the limit of protection.[1] Patients with diphtheria should be kept isolated until their throats are free from virulent bacilli. The bedroom,

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[1] With the von Behring vaccine (a mixture of toxin and antitoxin) immunity is not produced until after ten days, but it lasts at least a year.

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should be repeated. Laryngeal
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Next to alcohol, strychnin (1/120
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the purpose are warm normal salt
solution of boric acid. A solutio
of lime-water) or the following

Externally, hot or cold applications, whichever may be the more agreeable, are useful in relieving pain and soreness in the throat. In laryngeal cases tracheotomy or intubation, preferably the latter, should not be deferred if dyspnea becomes urgent.

Convalescence must be managed with special care on account of the tendency to sudden heart-failure. Anemia will require plenty of nourishing food, and such remedies as iron, arsenic, and cod-liver oil. Paralysis usually yields to strychnin and the use of massage and electricity.

RHEUMATIC FEVER

(Acute Articular Rheumatism; Inflammatory Rheumatism)

Definition.—An acute infectious disease, characterized by polyarthritis, irregular fever, acid sweats, and a marked tendency to endocarditis.

Etiology.—The disease is most common in the second and third decades. Males are more often attacked than females. It is most prevalent in cold, damp climates and in the changeable seasons of the year.

Exposure to cold and physical depression are predisposing factors. Disposition to recurrence is a characteristic feature.

The exciting cause is still unknown. According to one view the disease is an attenuated form of septicemia, due to streptococci or staphylococci; and, according to another, it is a specific infection due to one organism—the *Diplococcus rheumaticus* of Poynton and Payne.

Pathology.—The ligaments and the synovial membrane and its fringes are congested and swollen. The synovial sac is filled with turbid fluid. Suppuration, however, never occurs and almost invariably the arthritis ends in complete resolution, although the joints may be stiff for several weeks or months. Secondary inflammations, especially endocarditis, pericarditis, and pleurisy are frequently observed in fatal cases.

Symptoms.—The disease may follow an attack of acute tonsillitis, or it may set in at once with chilliness, fever, and inflammation of the joints. The joints involved are, as a rule, the larger ones, as the knees, ankles, elbows, and wrists. They are swollen, hot, painful, and tender, but only slightly reddened. The inflammation shows a marked tendency to flit from joint to joint, and to subside in one while attacking another. In severe cases the muscles also are painful and tender. Small subcutaneous nodules are sometimes found along the tendons and over the bony prominences. The fever in ordinary cases ranges between 102° and 103° F., and is very irregular. The perspiration is often copious and has an acid reaction and a peculiar sour odor. The appetite is lost, the tongue is coated, the bowels are constipated and urine is scanty and highly colored. Moderate leukocytosis is usually present, and as the disease progresses marked secondary anemia develops. Many *subacute cases* occur in which fewer joints are involved and the symptoms are of a mild type, but the course is often protracted.

The disease usually lasts from two to four weeks, but it may persist with alternate exacerbations and remissions for several months.

Complications.—Endocarditis, usually benign, is the most common complication, occurring in about 50 or 60 per cent. of all cases. The lesions most frequently affect the mitral valve. Pericarditis is also common, but less so than endocarditis. Myocarditis frequently accompanies endocarditis or pericarditis. Pleurisy may also occur. Tonsillitis is not rarely present at the onset of the disease.

In children chorea may precede, accompany, or follow rheumatic fever. Hyperpyrexia (108°–110° F.) is occasionally observed. In rare instances the high fever is accompanied by delirium, convulsions and coma and to this group of symptoms the term "cerebral rheumatism" has been applied. Certain cutaneous affections, especially purpura and various rms of erythema, are sometimes observed.

Diagnosis.—*Septic Arthritis.*—This may be recognized by its association with some other septic process and by the special tendency of the inflammation to end in suppuration, which is an extremely rare termination of rheumatic arthritis.

Gonorrheal rheumatism may be recognized by the history of gonorrheal infection; by the tendency of the inflammation to spread along the sheaths of the tendons; to remain stationary in joints primarily affected, and to attack joints rarely involved in rheumatism (intervertebral, sternoclavicular, temporomaxillary); by the stubborn character of the disease, and its resistance to salicylates.

Acute Arthritis Deformans.—In this disease certain joints are likely to be affected that usually escape in rheumatism, namely, the small joints of the hands, the intervertebral, sternoclavicular, and temporomaxillary; the inflammation rarely shifts from joint to joint; the temperature is usually between 99° and 101° F., but the pulse-rate is generally elevated out of proportion to the fever; there is marked tendency to muscular atrophy, especially of the interossei muscles, and to permanent deformity and fixation of the joints; and salicylates have little, if any, effect.

Gout.—This occurs later in life, usually involves the great toe, and lacks high fever, acid sweats, and the tendency to acute heart complications.

Prognosis.—In the great majority of cases rheumatic fever terminates in recovery. A very small number of patients die of exhaustion or some complication, such as endocarditis or hyperpyrexia, with grave nervous symptoms. The disease is very prone to relapse and to recur.

Treatment.—Absolute rest in a comfortable bed is essential, and, with the view of preventing permanent injury to the heart, this should be maintained for at least ten days or two weeks after the temperature has become normal and all the arthritic symptoms have subsided. The patient should wear a loose flannel night-dress and lie between blankets. Milk, eggs, and cereals are the most suitable articles

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interval between the doses sh
six hours. It is advisable to
days after the subsidence of
monium or sodium salt is not w
salophen or aspirin (1 dram da

Alkalies are useful adjuvants
from 15 to 20 grains of potassi
of sodium bicarbonate may b
hours, or until the urine is neutr

℞. Potassii citratis. . . .
Sodii salicylatis. . . . .
Aquæ menthæ piperitæ .
SIG.—A dessertspoonful every

Opium, in the form of Dov
hypodermically, is sometimes of
subduing restlessness, and pro
phenacetin, in moderate doses,
namia is marked, quinin (5 gra
Anemic patients are benefited by

Hyperpyrexia is best controlle
carditis and pericarditis rarely
The importance of prolonged res
becomes affected

Cloths kept moistened with methyl salicylate, saturated solution of magnesium sulphate, or lead-water and laudanum (ice-cold or hot), or ointments of salicylic acid are often serviceable.

℞.	Acidi salicylici	ʒiss
	Olei terebinthinæ	fʒj
	Adipis benzoinati q. s.	℥ij.

Sɪɢ.—Spread on lint and keep in place by means of a flannel binder.

No matter what local remedy is selected, it is highly important that the affected joints should be kept at complete rest. This may be accomplished by means of padded splints and a roller bandage.

Lingering swelling will often yield to an ointment of mercury and belladonna, with firm strapping of the articulation. Blisters are also useful. If the effusion is very great and persistent, it may be necessary to aspirate the joint.

For the stiffness of the joints massage, warm baths, and inunctions with an ointment of iodin will be found useful. The hot-air treatment also does good in some cases.

ERYSIPELAS

Definition.—An acute infectious disease excited by the Streptococcus pyogenes, and characterized by high fever and a peculiar inflammation of the skin and subcutaneous tissues.

Etiology.—The disease is somewhat contagious, and the poison can be carried in fomites. Certain families and certain individuals seem particularly predisposed. Puerperal women and wounded persons are very susceptible. Diseases which lower the vitality, especially Bright's disease, predispose. One attack does not protect against a recurrence, but rather favors it. Erysipelas was formerly divided into traumatic and idiopathic varieties; but the two are identical, and it is probable that in those cases in which there is no conspicuous wound there is a slight abrasion through which the infection gains admittance. The exciting cause of the disease is the Streptococcus pyogenes.

Pathology.—Erysipelas most frequently manifests itself on the face. The affected area is bright red in color, swollen, indurated, and sharply circumscribed. The various strata of the skin are infiltrated with serum, and leukocytes and streptococci are found in the lymph-spaces. In severe cases the inflammatory products are converted into pus, and abscesses form.

Symptoms.—The *period of incubation* is from three to seven days. Prodromes are sometimes present, and consist of slight fever, chilliness, malaise, and tingling in the part that is to be affected. In many cases the disease is ushered in suddenly with a chill, followed by pain in the head and limbs, and a high, irregular fever. The temperature may reach 104° or 105° F. in twenty-four hours. The pulse is full and rapid; the tongue is heavily coated; the appetite is lost; the bowels are constipated, and the urine is scanty and often slightly albuminous. In severe cases a typhoid condition, manifested by delirium, subsultus tendinum, a dry, brown tongue, etc., not rarely develops. There is usually a marked leukocytosis.

Local Phenomena.—The inflammation usually begins in the neighborhood of the nose, and spreads upward and laterally over the head to the neck, where it frequently stops. The affected part has a crimson hue; it is swollen and tense, and frequently ends in a sharply defined ridge, beyond which, however, projections can be felt advancing into the subcutaneous tissue. The surface of the inflamed patch is at first smooth and glazed, but later it is covered with minute vesicles or blebs. The patient complains of burning and tingling; the surrounding parts are extremely edematous, so that the features may be scarcely recognizable. In four or five days the redness begins to fade and the swelling to subside; desquamation follows; the general symptoms improve; and the fever falls by crisis or rapid lysis.

The average duration is from ten days to two weeks. In the so-called migratory cases, in which there is a successive involvement of new areas of integument, the disease may continue for several weeks. Relapses are very common.

Complications.—These are not common. Septicemia, ulcerative endocarditis, nephritis, arthritis, edema of the larynx, pneumonia, and meningitis are occasionally seen.

Diagonsis.—*Erythema.*—The absence of high fever, of marked swelling, and of an abrupt ridge will serve to distinguish erythema from erysipelas.

Acute Eczema.—The swelling is less marked; the itching is intense; the swelling and redness are not circumscribed, but shade gradually into healthy tissue; and there is no fever.

Prognosis.—In the robust the prognosis is favorable. In the aged, in alcoholic subjects, and in those suffering from chronic nephritis the prognosis must be guarded. Migratory erysipelas may kill by exhaustion. The average mortality is about 6 per cent.

Treatment.—As in other contagious diseases, isolation and disinfection are the most important prophylactic measures. Especially necessary is it to guard parturient and surgical patients from the contagion.

A supporting liquid diet should be given. Alcoholic stimulants are sometimes required. High fever is best controlled by cold sponging or the cold pack. Restlessness, delirium, and insomnia will call for applications of ice to the head, and perhaps the administration of morphin, chloral, or bromids.

Of the numerous special remedies recommended for erysipelas, the one which has enjoyed the most favor is the tincture of ferric chlorid (15 to 30 minims every three hours).

Local Treatment.—Among the numerous local applications recommended may be mentioned: Lotions of lead-water and laudanum, of carbolic acid (1:40), of picric acid (1:100), of magnesium sulphate (saturated solution), and of sodium salicylate (1:20). In the hands of the author ointments of ichthyol (20 per cent.) and of soluble silver (unguentum Credé) have proved most satisfactory.

The following combination often acts well:

℞.	Ichthyol.	gr. xxx
	Resorcinolis	ʒss
	Unguenti hydrargyri	ʒiv
	Adipis lanæ hydrosi.	ʒv.—M.

(Roswel Park.)

Local abscesses should be incised and treated antiseptically. Extension to the nose and throat will call for antiseptic sprays or washes.

PYOGENIC INFECTIONS

(Septicemia; Pyemia; Septicopyemia)

Definition.—Local or general non-specific infections caused by the entrance and growth within the body of the different pyogenic micro-organisms—most frequently the streptococcus pyogenes or the staphylococcus pyogenes aureus, but in some cases the Diplococcus pneumoniæ, B. coli communis, B. pyocyaneus, or Micrococcus gonorrhœæ. Mixed infections are common.

Etiology.—The infection may be acquired by inhalation or ingestion, but in the great majority of cases it is acquired by inoculation through a wound or an abraded surface. Susceptibility to infection is much enhanced by physical debility from any cause, although robust persons are by no means immune. In many cases pyogenic infection is a secondary or terminal event in the course of some other disease, such as chronic nephritis, arteriosclerosis, diabetes, etc. As a rule, the general infection is preceded by a local lesion, unobtrusive although it may be, as a furuncle, an infected Fallopian tube, a small tuberculous cavity, otitis media, cholecystitis, etc. Occasionally no portal of entry is discoverable, and to this group of cases the term *cryptogenetic septicemia* has been applied.

Pathology.—A general intoxication resulting from the absorption of soluble bacterial products formed only at the point of invasion is referred to as a *toxemia*. The group of symptoms associated with a general invasion of the blood and tissues by pyogenic organisms and therefore due to toxins elaborated in all parts of the body is described by the term *septicemia* (bacteriemia). If in the course of such a general invasion of the blood and tissues by pyogenic bacteria multiple secondary foci of suppuration occur, the condition becomes a *pyemia* (septicopyemia). These secondary foci include abscesses, ulcerative endocarditis, suppurative pylephlebitis, etc.

The lesion of a septicemia cannot be distinguished from those of a toxemia, but vary in each condition according to the character of the infecting bacterium. The spleen is usually more or less swollen, softened, and congested. The liver, heart, and kidneys present the appearances due to cloudy swelling and fatty degeneration. The liver frequently shows in addition small areas of necrosis. The lungs are congested and often present areas of pneumonic consolidation. Minute hemorrhages are sometimes observed beneath the skin and the serous membranes and within the substances of the various organs. In pyemia the lesions are similar, but in addition, secondary foci of suppuration, usually of embolic origin, are found in the lungs or in the territory of the general circulation.

Symptoms.—The symptoms vary with the severity and character of the infection, and those of toxemia differ from those of septicemia only in intensity. In mild cases there are chilliness, fever, reaching 101° or 102° F., headache, muscular pains, anorexia, a hot, dry skin, and restlessness. These symptoms usually subside rapidly after local treatment. In severe attacks the onset is marked by a sharp chill and a sudden rise of temperature to 104° F. or higher. The chills frequently occur at intervals and the temperature continues high with daily remissions or even intermissions. The usual concomitants of fever are present and vomiting and diarrhea

cases of malignant endocardit
chronic and for a long time n
temperature, an occasional ch
weakness.

Pyemia presents all the pheno
in addition the symptoms ari
suppuration in the lungs, kidn
Erythematous rashes are not
occurs in from two to three week

Diagnosis.—This is based upo
focus of infection and the gener
fever, sweats, leukocytosis, pallo
Malaria, typhoid fever, and acut
diseases most likely to come int
tion between a toxemia and a sep
the aid of blood cultures. The
certain by the discovery of secon

Prognosis.—This depends upo
the infection, the resistance of the
of removing the infectious foci. T
especially if due to the streptoc
Pyemia accompanied by ulcerat
suppurative pylephlebitis, or m
invariably fatal.

The results of serum treatment have not been very encouraging. Vaccines, especially those derived from a focus of infection in the individual under treatment (autogenous vaccines), are sometimes helpful; rarely so, however, except in cases which show a tendency to chronicity.

INFLUENZA

(La Grippe ; Catarrhal Fever ; Epidemic Catarrh)

Definition.—An acute infectious and contagious disease, characterized by fever, marked prostration, severe muscular pains, and catarrhal inflammation of the respiratory and gastro-intestinal tracts.

Etiology.—Influenza occurs endemically, epidemically, and often pandemically. The infection spreads with great rapidity, and may affect a large proportion of the population almost simultaneously. Outbreaks occur most frequently in colder weather and usually last about six or eight weeks. The disease is transmitted by contact and also by fomites. Doubtless "carriers" play an important part in disseminating it. No age is exempt, and one attack instead of conferring immunity predisposes to others. The exciting cause of influenza is the minute bacillus described by Pfeiffer in 1892, and which may be found in the sputum, blood, lungs, and other organs. Apparently, however, a disease clinically indistinguishable from influenza may be caused by the pneumococcus, streptococcus, or micrococcus catarrhalis.

Pathology.—Influenza does not often kill except by its complications. The latter are most frequently associated with the respiratory tract, and consist chiefly of pneumonia and its sequels, abscess of the lung, and pleurisy.

Symptoms.—The period of incubation is usually from two to four days. The disease begins abruptly with lassitude, chilliness, severe pain in the head and back, fever ranging between 102° and 104° F., and marked prostration. The catarrhal symptoms comprise injection of the eyes, sneezing, hoarseness, and harsh paroxysmal cough. In ordinary cases the temperature falls in three or four days by rapid lysis, but complications may prolong the attack for several weeks. As a rule, leukocytosis is absent. Relapses are common.

In some cases the catarrh of the respiratory tract is the chief feature; in others the gastro-intestinal tract is attacked, and the symptoms resemble cholera morbus; in a third group neuralgic pains in the head, back, and limbs are the most striking phenomena. A chronic pulmonary form has been described which is indistinguishable from tuberculosis except by bacteriologic tests.

Complications.—The most common are: lobar pneumonia, broncho-pneumonia, otitis media, conjunctivitis, neuritis, and functional disturbances of the heart. Occasionally endocarditis, myocarditis, thrombophlebitis, meningitis, or temporary insanity occurs.

Diagnosis.—*Acute Bronchitis.*—The fever is not so high; there is little or no prostration; and the pains in the head and back are not nearly so marked as in influenza.

Typhoid Fever.—The gradual onset, typical temperature-curve, epistaxis, Widal reaction, and rash will indicate typhoid fever.

Prognosis.—The prognosis is good, except in the aged and infirm in whom pneumonia and other complications are common.

Treatment.—Isolation, absolute rest in bed even in the mildest cases, sterilization of the secretions, regulation of the diet and careful nursing are the important general measures.

In mild cases a hot foot bath, some mild refrigerant, such as a spirit of nitrous ether or solution of ammonium acetate, and at night a dose of Dover's powder (5 to 10 grains) will usually suffice. If there be constipation, a few fractional doses of calomel may be given with advantage.

In more severe cases quinin (2 to 5 grains thrice daily) may be given throughout the attack. Pains are controlled to some extent by phenacetin with salicylates or benzoates.

The following combination is often useful:

℞. Acetphenetidini
Salophen
Sodii benzoatis. . . . . āā ʒj.—M.
Fiant chartulæ No. xij.
Sig.—One every three or four hours.

If the suffering is intense, morphin should be used hypodermically. Violent headache is treated best by small doses of phenacetin and the application of an ice-cap to the head. Heart-failure should be combated by alcohol, ammonia, and strychnin. Bronchial catarrh will require the remedies indicated in simple bronchitis.

MUMPS

(Infectious Parotitis; Epidemic Parotitis)

Definition.—An acute contagious disease, characterized by inflammation of the parotid and other salivary glands.

Etiology.—The disease occurs sporadically and epidemically. It is most frequently observed in young children, but unprotected adults are not exempt. Males are more susceptible than females. The disease is highly contagious, and the virus is probably contained in the saliva, but it has not been isolated. One attack usually confers immunity.

Pathology.—The lesions consist of congestion and edema of the parotid gland and periglandular tissues, and catarrhal inflammation of Stensen's duct. The process resolves without suppuration.

Symptoms.—The *period of incubation* is from ten days to three weeks. The disease is ushered in with chilliness, malaise, and moderate fever (101°–103° F.), followed by swelling of the parotid gland. The swelling is observed below and in front of the ear, is pyriform in shape, and has a doughy feel. The surrounding tissues are edematous, the submaxillary glands are often swollen, and the features may be distorted beyond recognition. The movements of the jaw are restricted and painful. The salivary secretion may be either increased or decreased. After an interval of a day or two the opposite gland usually becomes affected. The disease lasts a week or ten days and almost always ends in recovery.

Complications.—Orchitis, unilateral or bilateral, is common after puberty. It usually follows the parotitis but it may accompany it or even precede it. Atrophy of the testicle sometimes supervenes. In girls a transference of the inflammation to the vulva, mammary gland, or ovary is occasionally observed. Rare complications comprise maniacal delirium, otitis media, meningitis, nephritis, optic neuritis, pancreatitis (mild form), and chronic hypertrophy of the glands.

Treatment.—The patient should be kept in bed. Isolation should last three weeks from the onset of the disease. Mild aperients and refrigerants are useful. If the pain is severe, hot fomentations may be employed. In mild cases, covering the gland with cotton batting will suffice.

Orchitis will require rest, suspension of the affected gland, and the application of lead-water and laudanum or, better still, of an ointment of guaiacol (20 per cent.). After the tenderness has subsided, an ointment of mercury and belladonna will be found useful in reducing the swelling.

YELLOW FEVER

Definition. An acute infectious, endemic or epidemic disease, characterized by fever of one or two paroxysms, jaundice, albuminuria, and a marked tendency to hemorrhage, especially from the stomach.

Etiology.—The specific organism of yellow fever has not yet been isolated. Man is inoculated through the bites of a certain species of mosquito—*Stegomyia fasciata*—which serves as the intermediate host for the parasite. The mosquito is infected only by biting a yellow-fever patient during the first three days of the disease and cannot transmit the infection until a period of from twelve to twenty days has elapsed. The disease is not conveyed by fomites.

Yellow fever occurs endemically in certain tropical seaports (Rio de Janeiro, maritime towns of Central America), whence it occasionally spreads to temperate zones. The predisposing factors are those which are favorable to the growth of mosquitos—high temperature, surface drainage, and swampy soil. The colored race is less susceptible than the white. One attack usually confers immunity.

Pathology.—The tissues are stained yellow. The liver presents a mottled reddish-yellow (autumn-leaf) hue, and is the seat of extensive fatty degeneration. The kidneys usually show the lesions of acute hemorrhagic nephritis. The gastro-intestinal mucous membrane is swollen, congested, and frequently infiltrated with blood. The heart-muscle is pale and fatty.

Symptoms.—The *period of incubation* is from two to four days. The disease begins with a chill, followed by pain in the head, back, and limbs. The temperature rises rapidly until it reaches its maximum (103°–105° F.). The pulse is at first accelerated, but as the temperature rises it shows a marked tendency to fall, sometimes dropping in grave cases to 60 or even 50 a minute by the third day (Faget's law). The face is flushed; the conjunctivæ are injected; the pupils are small; the tongue is coated; the epigastrium is tender; the stomach is irritable and unretentive; the bowels are constipated; and the urine is scanty and often albuminous by the end of the first day.

Jaundice is rarely marked before the second or third day, although a slight icteroid tinge of the conjunctivæ is often noticeable within the first twenty-four hours. The first stage usually lasts from two to three days, and is followed by a rapid fall in the temperature and an improvement in all the symptoms (stage of calm or remission). At this time convalescence may begin or the patient may pass into the second stage.

The *second stage* is characterized by deep jaundice, persistent vomiting, vomiting of dark blood (*black vomit*), marked

The duration of the disease is fr

Diagnosis.—*Dengue.*—This

slow pulse with a rising tem

jaundice, or black vomit.

Acute Yellow Atrophy of the

diminution in the size of the live

cerebral symptoms, and the pr

in the urine will indicate acute

Malaria.—This may be distin

of the spleen, the multiple remissi

of the parasites, and by the abse

Prognosis.—This should alway

mortality in different epidemics is

individual cases high fever, a very

symptoms, black vomit, and su

favorable features.

Treatment.—"The spread of

effectually controlled by measure

of mosquitos and the protection

these insects."[1]

Absolute rest in a quiet, well-

nursing are essential.

call for dry cupping over the loins, alkaline diuretics, hot-air baths, and subcutaneous or rectal injections of warm saline solutions. Remedies have little effect upon the black vomit. Tincture of ferric chlorid, adrenalin solution (1:1000), and oil of turpentine have been recommended.

ACUTE GENERAL TUBERCULOSIS

(Acute Miliary Tuberculosis)

Definition.—An acute infectious disease excited by the tubercle bacillus, and characterized anatomically by the presence of miliary tubercles in many parts of the body.

Etiology.—General tuberculosis is always secondary to a more or less obvious primary focus of tuberculosis somewhere in the body (lungs, lymph-nodes, joints, etc.). The infection is transmitted by the blood stream. The disease occurs most frequently in early adult life and certain acute infections, such as measles and pertussis, and wasting diseases predispose to it.

Pathology.—All the organs may be uniformly infiltrated with discrete tubercles, but more commonly certain organs, like the brain and lungs, are more affected than others.

Symptoms.—The onset is gradual and characterized by anorexia, malaise, headache, increasing prostration, and fever. The temperature is moderately high (102°–104° F.), very irregular, and marked by evening exacerbations and morning remissions. The respirations are hurried and the pulse is rapid (140 to 150) and feeble. Cough may or may not be present. Profuse sweating is common. As the disease advances typhoid symptoms frequently develop—dry, brown tongue, muttering delirium, subsultus tendinum, carphologia and stupor. Tubercle bacilli are sometimes found in the cerebrospinal fluid, but rarely in the sputum or blood. The leukocyte count is usually low.

In the so-called *pulmonary form* there are: Dyspnea, rapid breathing (40 to 60 a minute), hard cough, mucopurulent and bloody expectoration, and cyanosis. Signs of con-

Pain, tenderness, abdominal di

Prognosis.—The disease is

duration is three or four weeks

Diagnosis.—Acute miliary tu

mistaken for *typhoid fever*, but

following differences:

Typhoid Fever
Epistaxis is common.
Temperature runs a regular course.
Diarrhea is frequent.
A roseolar eruption is generally present.
Respirations are rapid.
Pulse is rapid.
Cyanosis rarely marked.
Facial palsies are absent.
Widal reaction is present.
Tubercle bacilli are never present in the cerebrospinal fluid.

Treatment.—This is purely

consist of milk, eggs, and brot

Fever should be controlled by

of phenacetin. Severe cough

Etiology.—Whooping-cough occurs largely in epidemics and is most frequently observed in children, although unprotected adults are not entirely exempt. There is no doubt that the disease is contagious, especially in the catarrhal stage, that the infective agent is contained in the sputum, and that the infection may be transmitted by contact or by fomites. The exciting cause of the disease is probably the Bordet-Gengou bacillus which is found in large numbers among the cilia of the epithelial cells lining the air passages. One attack usually confers immunity.

Pathology.—There are no characteristic lesions. In fatal cases pulmonary complications such as bronchopneumonia, atelectasis, emphysema, etc., are usually present.

Symptoms.—There are three stages: (1) The catarrhal stage; (2) the paroxysmal stage; and (3) the stage of decline.

Catarrhal Stage.—The disease begins with the symptoms of coryza and bronchial catarrh—slight fever, sneezing, running from the nose, dry cough, and râles—but it does not respond to the ordinary remedies for catarrh, and after lasting one or two weeks passes into the paroxysmal stage.

Paroxysmal Stage.—The cough becomes more violent and paroxysmal. During the paroxysm the face is cyanosed, the eyes are injected, and the veins distended. The cough frequently induces vomiting, and, in severe cases, epistaxis or other hemorrhages. The close of the paroxysm is marked by a long-drawn, shrill, whooping inspiration due to spasm of the larynx.

The number of paroxysms, or "kinks," varies from ten or twelve to forty or fifty in the twenty-four hours. From the forcible propulsion of the tongue against the lower incisors, an ulcer is frequently formed on the frenum. The duration of this stage is three or four weeks.

Stage of Decline.—The paroxysms grow less frequent and less violent and finally cease. Protracted cases are followed by anemia and prostration.

During the entire course of the disease, which is usually from two to three months, a pronounced lymphocytic leukocytosis (20,000 to 40,000) is present.

Complications.—The chief complications are bronchopneumonia, acute emphysema, convulsions, and hemorrhage from the nose or into the conjunctiva. Paralysis from meningeal hemorrhage occasionally occurs. Severe cases are sometimes followed by cancrum oris, chronic bronchitis, or tuberculosis.

Prognosis.—Owing to the frequent occurrence of serious complications whooping-cough must be regarded as a dangerous disease, especially in infants.

Treatment.—Prophylaxis consists in isolation of the patient and the thorough disinfection of all articles that have been used by him. Quarantine should last until the cough ceases.

Fresh air, sunlight, protection from changes of weather, and a light but nutritious diet are essential. In some cases it may be desirable to keep the patient in his room, or even in bed, for the first few days, but ordinarily, if the weather is good, he need not be confined indoors. In advanced cases sea-air often acts most favorably.

Of the many special remedies advocated, those most worthy of confidence are belladonna (in ascending doses until constitutional effect is produced), antipyrin (about 1 grain for every year of the child's age, four times a day), quinin (10 grains a day at three years of age), bromoform (1 to 2 minims at two years of age), and sodium bromid (3 to 5 grains every three hours at two years of age). Chloral (3 grains at two years of age) may be given in severe cases at bedtime to secure sleep. Such a combination as the following may be useful:

℞.	Sodii bromidi	ʒiss
	Antipyrinæ	ʒi
	Glycerini	fʒii
	Aquæ menthæ piperitæ	q. s. ad f℥iij.—M.

SIG.—A teaspoonful every three hours for a child of three years.

The exact value of vaccine treatment has not been demonstrated; good results have been reported however, from injections into the buttocks every other day of from 50 million to 100 million bacilli.

Antiseptic and sedative sprays, when feasible, sometimes afford much relief; the best are the solution of hydrogen dioxid (1 to 6), menthol (5 per cent. in liquid paraffin), and resorcin (1 per cent. aqueous solution). An elastic abdominal belt is of some value in controlling the vomiting.

The child must be carefully guarded during convalescence, on account of the danger of catarrhal pneumonia. Tonics, especially quinin, iron, and cod-liver oil, are very useful at this period.

CHOLERA

(Asiatic Cholera; Epidemic Cholera)

Definition.—An acute infectious disease, generally epidemic, caused by the comma bacillus of Koch, and characterized by vomiting, excessive serous purging, painful cramps, and collapse.

Etiology.—Cholera is endemic in certain parts of India, and from time to time spreads into western countries. The exciting cause is the comma bacillus of Koch, a short, slightly curved, motile rod with a single flagellum. This organism is found abundantly in the intestinal discharges of choleraic patients, but not in the blood.

The disease always spreads along the lines of traffic, hence epidemics nearly always begin at the sea-coast and extend inland. Cholera is slightly, if at all, contagious. As in the case of typhoid fever human beings contract the disease by ingesting the germs, usually in water or on food. Flies are undoubtedly important factors in disseminating the infection to food. Laundresses and nurses, from contact with the evacuations, often acquire the disease. Epidemics are more frequent in summer than in winter. No age is exempt, but the aged are more susceptible than the young. The intemperate, the debilitated, and those suffering with gastro-intestinal catarrh are especially predisposed.

Pathology.—The body is shriveled; movements of the corpse are sometimes observed; rigor mortis is marked and prolonged. The tissues are dry, and the large veins and right side of the heart contain thick, dark blood. The serous cavities are empty and their surfaces sticky. The intestines contain more or less rice-water fluid, from which cultures of bacilli can be made.

The mucous membrane has a pinkish color and is often the seat of ecchymoses; the solitary and Peyer's glands are swollen. Frequently extensive desquamation of the epithelial lining is observed. The liver and kidneys are the seat of acute parenchymatous degeneration.

The symptoms of cholera are due to the absorption of a poison (endotoxin). Apparently normal epithelium prevents the absorption of the endotoxin contained in the bodies of the bacilli and set free by their destruction.

Symptoms.—The *period of incubation* is from two to five days. The severity of the symptoms varies considerably. In well-marked but favorable cases there are three stages: (1) Stage of invasion; (2) stage of collapse; (3) stage of reaction.

Stage of Invasion.—The disease usually begins with malaise, headache, diarrhea, rumbling noises in the intestines, and colic. Frequently these symptoms continue a few days and then subside; such cases are termed *cholerine,* and are as infectious as the fully developed disease.

Stage of Collapse.—The diarrhea grows more marked; the evacuations become copious, lose their feculent character, acquire a rice-water appearance, and are discharged forcibly but without pain. Vomiting soon develops, and the ejected material resembles that passed by the bowels. Thirst is unquenchable. Severe cramps seize the muscles of the calves of the legs, thighs, arms, and abdomen. The surface is cold and covered with a clammy sweat; the breath is cool; the temperature in the axilla ranges from 95° to 85° F., while in the rectum it may rise to 103° F. or more. The voice is husky and finally reduced to a whisper; the respirations are quickened; the pulse becomes more and more feeble; the body is livid and shriveled; the features are pinched and sometimes distorted; the eyes are frightfully sunken. The urine is more or less suppressed, and the little that is passed frequently contains albumin and sugar. Consciousness is usually retained until near the end, when coma sets in.

The duration of this stage is from a few hours to two days.

Stage of Reaction.—In this stage the symptoms gradually ameliorate, the stools become less frequent, the temperature returns to normal, the urine increases in amount and convalescence is soon established.

Occasionally, instead of convalescence, symptoms of a typhoid type develop (*cholera typhoid*), such as moderate fever, a dry brown tongue, muttering delirium, and coma. This condition, which is usually fatal, is probably the result of a secondary infection or of uremia.

Cholera Sicca.—This term has been applied to cases in which the intoxication is overwhelming and the patient dies within a few hours after the onset, even before the occurrence of diarrhea.

Complications and Sequelæ.—The chief complications are: nephritis, pneumonia, pleurisy, parotitis, ulceration of the cornea, croupous inflammation of the throat and fauces, abscesses, and local gangrene.

Diagnosis.—The differential diagnosis between Asiatic cholera and *cholera morbus* has already been considered (see p. 73).

Prognosis.—This depends largely upon the type. The mortality averages about 50 per cent. In the aged, very young, debilitated, and intemperate the disease is very fatal. In individual cases early collapse and a low surface temperature are unfavorable conditions. Most deaths occur within forty-eight hours after the onset.

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Precautionary measures p
isolation, absolute cleanliness
of excreta, soiled clothing, etc

The medicinal treatment of
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the reaction stage.

Prodromal Stage.—From th
the patient should go to bed a
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wise, aperients should be a
applied to the abdomen. If
may be given hypodermicall
subnitrate is perhaps the best

Algid Stage.—Intravenous i
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anhydremia and of restoring

warm applications, hot baths, gentle friction with anodyne liniments, and, above all, by intermittent chloroform inhalations. In suppression of urine the most promising measures are dry cupping over the loins and rectal and intravenous injections of saline solution.

Reaction Stage.—In this stage liquid foods in small quantities are permissible. Milk with lime-water, whey, thin gruels, albumin-water, and light broths are the most appropriate. The return to ordinary food should be effected very gradually.

DYSENTERY

The term dysentery is applied to a number of conditions characterized by abdominal pain, frequent diarrheal discharges containing mucus and blood, and tenesmus. Anatomically, there may be catarrhal, ulcerative or pseudo-membranous changes in the intestine, especially in the colon. The excitants of dysentery may be chemical irritants, various bacteria, or certain protozoa (Ameba coli, Balantidium coli). Two specific varieties are recognized: (1) Bacillary, and (2) amebic.

BACILLARY DYSENTERY

Etiology.—Bacillary dysentery is caused by the Bacillus dysenteriæ, described by Shiga, Flexner, Kruse, and others, and of which there are several strains. The disease is especially prevalent in Japan, the Philippines, and other tropical countries, but it frequently occurs elsewhere both sporadically and in epidemics. As in typhoid fever, the infection is transmitted chiefly by contaminated food and drink. Doubtless flies often aid in its dissemination. Overcrowding and unsanitary conditions in general favor its spread, hence it has prevailed extensively at times in camps, jails, etc. Lowered vitality from any cause is a predisposing factor, and in consequence dysentery is not an uncommon secondary or terminal event in many wasting diseases. The summer diarrheas of children may also be associated with the dysentery bacillus.

Pathology.—The disease is usually acute, but it may be chronic. The changes predominate in the large bowel and in acute cases usually consist of hyperemia, swelling of the mucosa, enlargement of the solitary follicles, and superficial erosions. The lymph-glands are often enlarged. In some cases false membrane is formed on the surface of the bowel (*diphtheritic dysentery*), and in others more or less widespread gangrene occurs. In chronic cases the mucous membrane is thickened and reddened and shows superficial erosions, but rarely any pronounced ulceration.

Symptoms.—The disease begins with colic and diarrhea. Within twenty-four hours tenesmus develops and the stools become mucous and bloody. In some cases shreds of false membrane are passed. The temperature rises usually to between 101° and 103° F., the tongue is coated, the appetite is lost, the abdomen is tender and somewhat excavated, and vomiting sometimes supervenes. Weakness and emaciation rapidly develop. Toxic symptoms—muscular pains, delirium and stupor—are not uncommon in serious infections. The blood serum yields an agglutination reaction with dysentery bacilli of the type isolated from the stools. In fatal cases death usually occurs after two or three weeks, but it may occur within three or four days. In favorable cases the duration is from ten days to three or four weeks. Occasionally the disease loses its acute character and continues with remissions and exacerbations for an indefinite period.

Complications.—Peritonitis, usually circumscribed and due to extension of the inflammation through the bowel, is not uncommon. Inflammation of other serous membranes, arthritis, peripheral neuritis, and parotitis are sometimes observed.

Diagnosis.—The diagnosis of dysentery can usually be made without difficulty from a study of the general symptoms. The determination of the exact nature of the infection, however, must rest upon the serum agglutination tests and the isolation of the bacillus from the stools. In *acute catarrhal enteritis* tenesmus is absent; the stools are not bloody and mucous, nor are they so frequent or so small. *Intussusception* may be distinguished by the abrupt onset, persistent vomiting, sausage-shaped abdominal tumor, and absence of fever, at least at the outset.

Prognosis.—This depends upon the severity of the attack and the resistance of the individual. The more marked the toxic symptoms are the more unfavorable is the outlook. In some epidemics the mortality has reached 40 per cent.

Treatment.—The prophylactic measures adopted in typhoid fever and cholera are applicable in dysentery. Absolute rest in bed is imperative. In acute cases the diet should consist of milk diluted with barley-water, peptonized milk, infant foods, egg albumin, chicken broth, and milk toast. In chronic cases, soft boiled eggs, pulled bread, steamed rice, oysters, tender meats, wine-jelly, and custard may be allowed. An unirritating purgative (magnesium sulphate, calomel, or castor oil) is indicated at the onset. Subsequently opium should be given to inhibit peristalsis and to relieve the tenesmus. It may be given hypodermically in the form of morphin, or by the stomach or bowel. Turpentine stupes or sinapisms over the abdomen are advisable. Tenesmus may also be relieved by ice suppositories, iodoform suppositories, injections of warm mucilage of starch (1 ounce) or of cocain solution (10 minims of a 4 per cent. solution), or irrigations with warm normal saline solution. In the chronic form irrigations with solutions of silver nitrate (10 to 20 grains to the pint) are effective. These should be given every day, the fluid being introduced very gently by means of a fountain syringe. Internally, bismuth subnitrate (30 to 40 grains) with antiseptics, like salol (5 grains) or benzonaphthol (5 grains), is useful. Considerable benefit may also be derived from serum therapy. The serum should be polyvalent or, if feasible, one corresponding to the type of infection present. From 50 to 100 c.c. may be given every six to twelve hours. For collapse stimulants and subcutaneous injections of saline solution may be used.

AMEBIC DYSENTERY

Etiology.—This form of dysentery prevails chiefly in the tropics, but sporadic cases and even small epidemics not very rarely occur in temperate regions. The exciting cause of the disease is the ameba coli (*Entameba histolytica*), a unicellular protoplasmic mass about 15 to 30 μ in diameter, containing a nucleus and one or more vacuoles. The infection is usually transmitted through drinking water.

Pathology.—The lesions are found mainly in the large intestine. At first the mucous membrane is hyperemic and edematous. Later localized areas of cellular infiltration appear in the form of hemispheric elevations and these ultimately break down, leaving deep ulcers with infiltrated undermined edges. In advanced cases the whole intestinal wall is much thickened. The amebæ are found in the dejections, and at the base of the ulcers, in the adjacent lymph-vessels, and sometimes in the blood-vessels, whence they are frequently (20 per cent. of the cases) conveyed to the liver where they cause colliquative necrosis and the so-called amebic abscess.

Symptoms.—Mild attacks are not rarely observed, in which for weeks or months the only symptoms are indigestion, colicky pains, and slight diarrhea. The diagnosis rests upon the discovery of the parasites in the stools. Recovery is the rule, but the condition may become acute or may slowly pass into a chronic phase. In other cases the infection is acute, and marked by fever, abdominal pain, vomiting, tenesmus, mucus and blood discharges, great weakness and emaciation. Death may result from exhaustion, or the condition may gradually become chronic. Irrespective of the mode of onset, the disease shows a marked tendency to assume a chronic form. It is then characterized by continuous or intermittent diarrhea; the passage of mucus, blood, and perhaps pus in the stools; more or less abdominal discomfort; afternoon fever; and, ultimately, marked anemia and wasting. Tenesmus may not be pronounced. Uncomplicated cases usually yield to treatment, but relapses are extremely common. Death is most frequently due to *hepatic* or *hepatopulmonary abscess.* Other *complications* are perforation of the bowel, peritonitis from extension of the ulcerating process, intestinal hemorrhage, and cicatricial stenosis of the bowel.

Diagnosis.—Unlike the amebic form, *bacillary dysentery* is usually acute, is rarely associated with pronounced ulceration of the bowel or hepatic abscess, and is frequently productive of a general intoxication. *Syphilis* and *carcinoma* of *the rectum* must always be considered in the differential diagnosis.

Treatment.—The same general measures should be followed as are adopted in bacillary dysentery. Ipecacuanha has been shown to exert a specific effect and should always be given a thorough trial. The drug may be given in pills heavily coated with salol (40 grains the first night and reduced 10 grains each night until only 10 grains are given) or, better, subcutaneously in the form of emetin hydrochlorid (½ to 1 grain in 20 minims of salt solution, two or three times a day). Irrigation of the bowel is efficacious. It should be done once a day by means of a fountain-syringe, the tube being inserted high up and the fluid (a quart to two quarts) retained, if possible, for fifteen minutes. The best solutions are, perhaps, quinin (1 to 5000 gradually increased to 1 to 1000) and silver nitrate (1 to 2000 gradually increased to 1 to 500). If the rectum is very irritable, it is advisable to inject a small quantity of cocain solution (4 per cent.) before introducing the irrigator. In intractable cases recourse may be had to colostomy or appendicostomy and irrigation of the colon through the wound.

TETANUS

(Lockjaw)

Definition.—An acute infectious disease excited by a special bacillus, and characterized by painful tonic spasms of the voluntary muscles.

Etiology.—The exciting cause is the *Bacillus tetani*, a motile, spore-bearing, anaërobic rod, multiplying in garden-earth,

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to the spinal cord where
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rigidity in the muscles of
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Diagnosis.—In *strychni*

death usually resulting within a week from heart-failure, asthenia, or asphyxia. Cases developing after a long period of incubation and not characterized by violent seizures not infrequently end in recovery. The average mortality is about 60 per cent.

Treatment.—The most important safeguard against the occurrence of tetanus is the thorough cleansing and disinfection of all wounds. When there is ground for suspicion that tetanus may develop, as in wounds contaminated by garden earth, street dirt, or stable refuse, the local treatment should be supplemented by prophylactic injections of tetanus antitoxin (1500 units). The injections should be made deeply into the muscles in the neighborhood of the wound, and should be repeated at the end of a week. In the fully developed disease antitoxin is of much less value, but it should be tried. Injections should be made daily, under chloroform anesthesia, into the subdural space of the cord (3000 to 5000 units), into the motor nerves leading from the wounded part (500 to 1000 units) and into one of the large veins (10,000 units). The drugs most effective in subduing the convulsions are the bromids and chloral. These should be given in large doses. Morphin is a useful adjuvant. Inhalations of chloroform afford temporary relief. The patient should be kept absolutely quiet and protected from cold. The administration of nutriment in liberal quantities (by nasal tube if necessary) is of the utmost importance. Continuous enteroclysis is often advisable.

EPIDEMIC POLIOMYELITIS

(Acute Anterior Poliomyelitis; Acute Infantile Spinal Paralysis)

Definition.—An acute infectious disease, occurring chiefly in young children, characterized anatomically by inflammation of the gray matter of the spinal cord with destruction of the nerve-cells in the anterior horns, and manifested clinically by febrile disturbances and rapid atrophic paralysis of various muscles.

Etiology.—The disease may be sporadic or epidemic. It is more prevalent in cold than in warm countries. Most cases occur in the summer months. Children below the age of six years are especially susceptible, but adults may be attacked. The affection is contagious, though probably not highly so, and doubtless is often transmitted by healthy persons ("carriers") who have been in contact with the sick. The researches of Flexner and Noguchi have demonstrated that the causative agent is a minute anaërobic organism and that the infection atrium is the nasal mucosa.

Pathology.—The changes are most frequently found in the lumbar segments of the cord and are especially pronounced in the anterior horns of gray matter, but not rarely the medulla, pons, and cerebrum are also affected. The meninges often share in the inflammatory process. In early cases microscopic examination reveals distention of the blood-vessels, cellular infiltration, and degenerative changes in the ganglion cells. In long-standing cases the lesions consist in atrophy of the anterior horns, loss of ganglion cells, and overgrowth of neuroglia.

The motor nerve-fibers corresponding to the diseased segments and the muscles innervated by those fibers are also the seat of degenerative changes.

Symptoms.—The onset is marked by moderate fever (101°–103° F.), restlessness, headache, pain in the back and limbs, and muscular soreness. Less frequently there is vomiting or diarrhea, and occasionally convulsions occur. The mind is usually clear. In the course of a day or two a flaccid paralysis makes its appearance, all four extremities and the trunk, the lower extremities, one limb, or, perhaps, only a group of muscles being involved. The legs are especially prone to suffer. The paralysis reaches its maximum in a few hours or days, and then begins to improve, in many cases only a small amount remaining at the end of a few weeks or months. Complete recovery is sometimes observed. The constitutional symptoms soon pass off; sensation remains intact; the sphincters are not disturbed, and there is no tendency to bed-sores. The muscles showing decided paralysis lose their tendon reflexes, rapidly waste, and soon yield the reaction of degeneration. Permanent deformity often ensues from the retardation of growth in the paralyzed members and the overcontraction of the unantagonized muscles.

Varieties.—Apart from the common *spinal* form, there are (1) *abortive* cases, in which the constitutional disturbance is unattended by paralysis and complete recovery occurs in a few days; (2) *meningitic* cases, in which the early symptoms simulate closely those of epidemic cerebrospinal meningitis; (3) *bulbar* cases, in which the nuclear centers in the medulla oblongata are involved; and *polyneuritic* cases in which pain in the limbs and general hyperesthesia are for a few days the most conspicuous features.

Diagnosis.—Except in epidemics the diagnosis is not possible before the appearance of paralysis. In *multiple neuritis* the paralysis develops more gradually, is more marked in the distal than in proximal parts of the limbs, and is symmetric, and, moreover, the sensory disturbances are more lasting. *Myelitis* may be distinguished by the presence of anesthesia, paralysis of the bladder and rectum, and the tendency to bed-sores. The early occurrence of flaccid paralysis and the absence of cocci in the cerebrospinal fluid will distinguish the meningitic type from *epidemic cerebrospinal meningitis.* The *cerebral paralyses of childhood* are spastic, attended by exaggerated reflexes, and not followed by rapid wasting.

Prognosis.—Unless the initial symptoms are very severe or the muscles of respiration are affected, the prognosis as regards life is good. The death-rate has ranged between 5 and 30 per cent. In all cases much of the paralysis disappears, and occasionally the improvement is so marked that the usefulness of the members is not seriously impaired.

Treatment.—During the acute stage the patient should be isolated and confined to bed. Mild laxatives and febrifuges may be used with some advantage. Hexamethylenamin (2 or

3 grains, every two hours during the acute stage) has been recommended by Dana and others for the purpose of sterilizing the cerebrospinal fluid. Aspirin or morphin may be necessary for the relief of pain. Warm baths and lumbar puncture are also worthy of trial. The affected limbs should be wrapped in cotton-wool. In the course of two or three weeks, if the acute features have entirely subsided, the use of massage and electricity should be begun. The treatment of the latter stages is chiefly surgical, and has for its object the prevention or correction of deformities.

DENGUE

(Breakbone Fever; Dandy Fever)

Definition.—An acute infectious disease, characterized by pains in the muscles and joints, a variable rash, and a febrile course of two paroxysms.

Etiology.—Dengue is confined almost entirely to hot climates. Although it occurs in epidemics, it is probably not contagious, but transmitted through infected mosquitoes.

Symptoms.—The *period of incubation* is from three to five days. The invasion is usually sudden, and is attended with lassitude, chilliness, headache, intense pain in the muscles and joints, and high fever. The latter rises rapidly, often reaching a maximum of 104° to 105° F. in a few hours. The skin and conjunctivæ are congested; the pulse is accelerated (100–130); the urine is scanty; the superficial lymph-glands are enlarged; and the joints are painful, tender, and slightly swollen; mild delirium is sometimes noted. In three or four days sweating occurs; the temperature falls; and the pains abate, leaving the patient comparatively comfortable, but extremely weak. This remission lasts one or two days, and is followed by a second paroxysm, in which all the original symptoms return, though with less severity and for a shorter period. A roseolar eruption usually accompanies the second elevation of temperature. It lasts one or two days and is followed by desquamation.

Complications are rare and recovery is the rule. Hemorrhages from the mucous membranes occasionally occur. Convalescence is often slow, the soreness in the joints persisting for a long time.

Diagnosis.—*Acute Rheumatism.*—This disease runs a more protracted course, and lacks the paroxysmal character and the eruption of dengue.

Treatment.—There is no specific remedy. A mercurial aperient should be given at the onset. The pains are best relieved by phenacetin, salicylates, and morphin. The diet should be liquid and sustaining.

HYDROPHOBIA

(Rabies; Lyssa)

Definition.—A specific infectious disease of certain carnivorous animals, especially dogs and wolves, communicated to man by direct inoculation, and characterized by slight fever, intense spasm of the muscles of the throat, delirium, paralysis, and coma.

Etiology.—Rabies invariably results from the bite of a rabid animal, usually a dog. In the animal the disease is characterized by depression of spirits, loss of appetite, followed by excitement, aimless roving, a morbid desire to bite, and finally by paralysis and death from exhaustion. The poison is contained in the central nervous system and secretions, especially the saliva. Bites on the face and on exposed parts are particularly liable to be followed by infection.

Pathology.—The bacteriology is obscure. Microscopically, accumulations of embryonic cells are found in the cord and bulb surrounding the motor nerve-cells or displacing them (rabic tubercles of Babes). Still more important from a diagnostic standpoint, hyaline bodies, probably protozoa, are found in the cells of the cerebellar cortex, horn of Ammon, and spinal ganglia (Negri bodies).

Symptoms.—The *period of incubation* is usually from three weeks to three months, rarely as long as twelve months. The *onset* is characterized by slight fever, anxiety, depression, restlessness, and pain in the wound or cicatrix. In about a day symptoms of the *convulsive stage* appear. These consist in great difficulty in swallowing, severe clonic spasms of the laryngeal muscles, salivation, general hyperesthesia, tetanoid seizures, hallucinatory delirium, and prostration. Anything that excites the swallowing reflex, such as the sight of water, may bring on the painful spasm of the throat muscles. In the course of two or three days, if the patient does not die from exhaustion or heart-failure, the *paralytic stage* supervenes, in which the convulsions and delirium give way to ascending paralysis and unconsciousness.

Diagnosis.—An hysterical condition, known as *pseudohydrophobia*, is not uncommonly observed in neurotic persons who have been bitten by non-rabic dogs. It may simulate true hydrophobia, but there is no fever, the symptoms last longer than those of rabies, and the patient often barks like a dog, tries to bite, and becomes very emotional.

Prognosis.—Once developed, the disease is invariably fatal.

Treatment.—Suspicious bites should be thoroughly disinfected and cauterized with caustic potash or strong carbolic acid and treated as open wounds. As soon as possible the patient should be given the benefit of Pasteur's immunizing treatment, which consists in a series of inoculations with properly prepared spinal cords from artificially infected rabbits. The mortality from rabies among patients promptly immunized is less than 1 per cent. The treatment of the attack is purely palliative. An attempt should be made to maintain nutrition by rectal alimentation and to control the convulsive paroxysms by injections of morphin and inhalations of chloroform.

LEPROSY

(Lepra; Elephantiasis Græcorum)

Definition.—A chronic infectious disease caused by the Bacillus lepræ, and characterized by granulomatous formations in various parts of the body, more particularly in the skin, mucous membranes, and nerves.

Etiology.—Leprosy exists to a greater or less extent in all parts of the world, but it is especially prevalent in India, China, Japan, and other oriental countries. In the United States the chief foci are in Louisiana and California. The disease is only mildly contagious. The exciting cause is the Bacillus lepræ, which resembles the tubercle bacillus and which is found in the specific lesions and in the secretions of the nose and mouth.

Pathology.—The bacillus excites a chronic proliferative reaction, resulting in the formation of a granuloma, somewhat like that observed in tuberculosis and syphilis. Caseation, however, does not occur, but necrosis of the individual cells ensues and slowly advances to the stage of ulceration.

Symptoms.—The *period of incubation* may be but a few weeks or as long as three or four years. Prodromal symptoms, consisting of headache, malaise, irregular attacks of fever, and pains in the joints, are sometimes observed. Two clinical forms are recognized, the nodular (tubercular) and the anesthetic, but very often the manifestations are mixed. The *nodular type* is characterized by the development of large or small nodular elevations of a dusky-red color, in various parts of the body, especially on the face, the extensor surfaces of the elbows and knees, and the hands. After a time these break down, forming ulcers which do not readily heal, or they are transformed into dense cicatricial tissue, causing unsightly deformities. The mucous membranes of the eyes, nose, and throat may also be involved. The *anesthetic type* is manifested by lancinating pains and areas of hyperesthesia, followed by the appearance of yellowish macules, which spread peripherally and eventually become anesthetic. Sooner or later trophic changes occur. These comprise milk-white patches (lepra alba), areas of brown pigmentation, bullous eruptions, loss of hair, muscular atrophy, mutilating ulcers, and a disappearance of the phalanges one by one, through disintegration and absorption (lepra mutilans). In advanced cases the skin of the face is irregularly thickened and nodular (leontiasis), the ears are leathery, the facial hair is lost, the eyelids are everted, and the hands and feet are crippled and deformed. The diagnosis is made certain by the detection of the lepra bacilli in an excised nodule or in the nasal secretion. The disease is very chronic, often lasting twenty years or more, and occasionally it is arrested.

Treatment.—Segregation is advisable in all cases. The patient's general nutrition should be improved by wholesome, digestible food, personal cleanliness, frequent warm baths, and tonics. Of the many special remedies recommended, chaulmoogra oil (doses of 5 minims, very gradually increased to 200 minims, three times a day) seems to be the only one possessing real merit.

PLAGUE

Definition.—Plague is an acute infectious disease, excited by the *Bacillus pestis bubonicæ,* characterized by high fever, extreme prostration, inflammation of the lymph-glands (buboes), and often by pustules, pneumonia, and hemorrhages.

Etiology.—The plague bacillus, which is a short, encapsulated, non-motile, non-spore-bearing organism, is found in all the fluids and organs of the infected body. Transmission of the disease may occur directly through the entrance of infected material into some abrasion of the skin or a mucous membrane, but in the large majority of cases it probably occurs indirectly through the bite of the rat-flea, this insect conveying the infection from the rat, an animal particularly susceptible to plague. The disease may occur in any climate and in any season, but it flourishes best in the tropics and in warm weather. Persons of any age may be attacked. Overcrowding and bad sanitary conditions favor outbreaks.

Pathology.—Three forms of buboes may be recognized: *Primary buboes,* which develop near the point of inoculation and which are characterized by marked periglandular edema;

secondary buboes, which occur in the immediate neighborhood of the primary lesion and are produced by infection through the lymph vessels; and *tertiary buboes*, which occur in all parts of the body and are caused by infection from the blood. Suppuration rarely occurs in the last two varieties, but is common in primary buboes. The skin, mucous membranes, and serous membranes are often studded with punctate hemorrhages. The lungs are congested and frequently show areas of bronchopneumonia. The heart is flabby and dilated. The spleen is enlarged and congested. The liver and kidneys are swollen, hyperemic, and show cloudy swelling.

Varieties.—Four forms are generally described: bubonic, pneumonic, septicemic, and abortive or ambulatory.

Symptoms.—The *period of incubation* varies from two to ten days.

Bubonic Plague.—The onset is usually sudden, with chilliness, headache, general pains, vomiting, and great prostration. The fever is high and irregular. The general appearance is often suggestive; the eyes are bright, the expression is anxious, the nostrils are dilated, the body is flexed. The buboes usually appear early or within a few hours of the onset, and are most frequent in the groin, femoral region or axilla. Suppuration takes place in the second week and is not rarely followed by gangrene. The pulse is frequent and feeble, the respirations are hurried, and the urine is scanty. Delirium is common. Pustules sometimes appear on the body and in many cases multiple hemorrhages are noted. In the majority of cases death occurs before the buboes suppurate.

Pneumonic Plague.—In this form severe pneumonia, usually of the lobular type, is the important feature. The general symptoms are similar to those of bubonic plague, but primary buboes are usually absent. Death occurs, as a rule, within the first week.

Septicemic Plague.—In this comparatively rare form no primary point of infection can be detected. However, all the lymph-glands are more or less enlarged, the systemic

the type of disease, and the
individual case. The death-r

Prophylaxis and Treatmen guards against the spread sick, rigid quarantine, thorou discharges and all articles tha the discharges, and good ge prevention of overcrowding, th garbage, and the destruction oculation with sterilized culture of trial. Treatment of the d Lustig's serum seems to have s

GLANDUL

This is an infectious, probably con hood, characterized by chilliness, h fever, slight reddening of the thro flammatory swelling of the lymph- cervical glands, posterior to or jus The glands rarely suppurate and com has been noted. The duration varie treatment is essentially symptomatic

ROCKY MOUNTAI

This

and ankles, and then spreads over the body. In the course of a few days the macules become definitely petechial. Gangrene of the skin in certain parts and general edema are sometimes observed in severe cases. The average mortality in some districts (Bitter Root Valley) is 90 per cent., but in Idaho it is less than 5 per cent. Death occurs most frequently within the first ten days. The treatment is altogether symptomatic.

KALA-AZAR

(Tropical Splenomegaly; Dum-dum Fever)

Kala-azar is a chronic, specific, infectious disease, prevailing especially in India and certain other regions of the Orient. It is caused by the Leishmania donovani, a round or oval body about the size of a blood platelet, which is probably conveyed by the bed-bug or some other biting insect.

The disease begins with high fever and rapid enlargement of the liver and spleen. The fever usually subsides in from two to six weeks, but recurrences are the rule and finally there may be a low continued fever. As the disease progresses marked weakness, emaciation, and anemia supervene, the skin acquires a grayish color, and general edema frequently develops. The spleen extends below the navel in three-fourths of the cases. The mortality is said to be 96 per cent. In addition to general hygienic measures, quinin in large doses, and arsenic are believed to be of service in treatment.

MALTA FEVER

(Mediterranean Fever)

Definition.—An infectious disease of long duration, excited by the *Micrococcus melitensis* and characterized by periods of fever, alternating with periods of normal temperature, profuse sweats, neuralgic pains, progressive weakness, and anemia.

Etiology.—While the disease is especially prevalent in Malta, it has frequently been reported from other tropical and subtropical countries. It has been proved conclusively that the large majority of cases are due to the use of milk from infected goats. Infection through contaminated food other than goats' milk, through the inhalation of dust containing the micrococci, or through the bites of insects appears to be comparatively rare. The organism is found in the blood and leaves the body in the urine, feces, and milk. The period of incubation is about two weeks.

Symptoms.—The most striking symptom is the fever. This usually rises gradually to a maximum of 103° or 104° F., runs an irregular remittent course for from one to five weeks, and then gradually drops to normal, where it remains several days, when a relapse occurs. This sequence observed.

Prognosis and Treatment.—The and ultimate recovery. The mortal treatment is entirely symptomatic. measure.

SLEEPING

(Trypano

Definition.—An infectious disease, excited by the *Trypanosoma gambien* the lymph-glands, moderate fever, p increasing lethargy, and, finally, deat

Etiology and Pathology.—The caus *soma gambiense*, a minute, flagellat entrance to the body through the bit The organism is found in the lymp fluid. The symptoms of the disease lation of lymphocytes in the perivas cord, secondary degeneration of the roglia (chronic meningo-encephalomy

Symptoms.—The course of the dis stage, which lasts from a few mont symptoms are enlargement of the trypanosomes in the blood and lympha lasts several months and is marked b

WORM INFECTION—HELMINTHIASIS

TAPEWORM OR CESTODE INFECTION

Varieties.—Tænia solium; Tænia saginata; Dibothriocephalus latus; Hymenolepis nana; Tænia echinococcus.

Tænia Solium (*Pork-measle Tapeworm*).—This worm exists in the larval state (*Cysticercus cellulosæ*) in the hog, which becomes infected by eating food contaminated with the eggs of the parasite derived from human feces. Man becomes infected by eating raw or rare pork containing the larvæ. The mature worm is from 2 to 6 yards in length. The head has a double row of hooklets, and is attached to the body by a thread-like neck. The uterus in the gravid segments has from 15 to 35 lateral branches on each side of its median stem. When ova gain access to the stomach of man through the pylorus, as in vomiting, or by the ingestion of contaminated food, they produce embryos which pass through the walls of the alimentary canal, effect a lodgment in the muscles or organs, and there develop into bladder-like larvæ (*cystercercosis*). This parasite is rare in America.

Tænia Saginata (*Fat Tapeworm*).—The larval form occurs in the ox, which becomes infected by eating food containing embryos derived from human feces. Man becomes infected by eating undercooked or undercured beef. The mature worm is from 5 to 10 yards in length. The head is without hooklets, and the median stem of the uterus gives off on each side from 15 to 35 slender branches. Tænia saginata is the most common tapeworm in North America and certain parts of Europe.

Dibothriocephalus Latus (*Broad Tapeworm*).—The larval form of this parasite is found in certain fresh-water fish.

Man is infected by eating uncooked fish. The mature worm is of yellowish-brown color, and is from 2 to 10 yards in length. The head is flat and without hooklets; the segments are usually broader than long; the sexual opening is in the middle of the flat surface of the segment, and the uterus is in the form of a roset. This worm is common in Northern Europe, but rare in America.

Hymenolepis Nana (*Dwarf Tapeworm*).—This parasite develops without any intermediate host. Man is infected probably through food contaminated with eggs derived from the feces of infected rats or other human beings. The adult worm is only a few millimeters long. The head is small and provided with a single circle of hooklets. Great numbers are usually present. The geographic distribution of the parasite is extensive.

Tænia Echinococcus.—The adult worm occurs in the intestine of the dog. In man the parasite appears only in the larval state (see Hydatid Cysts of the Liver).

Symptoms.—In many cases of cestode infection there are no subjective symptoms. Some patients, however, present digestive disturbances, bulimia, colicky pains, anal pruritus, emaciation, anemia, and certain reflex phenomena—vertigo, palpitation, itching of the nose, and choreiform movements. The diagnosis rests on the detection of segments or eggs in the stools. Infection with Dibothriocephalus latus may be the cause of severe anemia, which is attributed to a toxin produced by the parasite.

Treatment.—By way of preparatory treatment it is advisable to restrict the diet for a day or two to liquids, and to empty the bowel as completely as possible by saline purges. The best teniafuges are oleoresin of aspidium (½ to 2 drams), pelletierin (5 to 10 grains), and pumpkin seed (2 to 4 ounces).

℞. Oleoresinæ aspidii	f℥ss
Pulveris acaciæ et sacchari	āā q.s.
Aquæ cinnamomi	q.s. ad f℥ij.—M.

Sɪɢ.—One tablespoonful, to be repeated if necessary.

A full dose of Epsom salts or compound jalap powder should be given a few hours after the teniafuge. The treatment is successful only when the head of the worm is passed.

ROUND-WORM OR NEMATODE INFECTION

Ascaris Lumbricoides (*Eel-worm or Common Round-worm*). —Eel-worms develop from eggs which have entered the digestive tract through contaminated water or food. No intermediate host is required. They are of a grayish or pinkish color, and in form resemble earthworms. They live in the small intestine, but not rarely, especially in febrile conditions, they migrate, entering the stomach, bile-ducts, respiratory tract, or urinary passages, or perforating into the abdominal cavity through an intestinal ulcer.

Symptoms.—Symptoms are often absent. Sometimes there are dyspepsia, voracious appetite, colicky pains, mucous stools, anal pruritus, anemia, and reflex nervous phenomena—night-terrors, grinding of the teeth, disturbances of vision, choreiform movements, and convulsions.

Treatment.—Santonin (¼ to ½ grain); wormseed oil (10 minims in capsule or on sugar); and fluidextract of spigelia (1 to 2 fluidrams) are efficient remedies. The anthelmintic should be followed by a purge.

℞. Santonini	gr. vj
Hydrargyri chloridi mitis	gr. vj
Sacchari	gr. xxiv.—M.

Fiant chartulæ No. xii.

SIG.—One powder morning and evening (Starr).

Oxyuris Vermicularis (*Pin-worm; Seat-worm*).—Pin-worms are small white worms, measuring from one-eighth to one-half inch in length. They are most frequent in children, but may occur at any age. Infection occurs directly through the eggs, which are conveyed to the mouth on the fingers or enter the stomach through contaminated food. The adult worms are most numerous in the cecum. The females migrate to the rectum, where they deposit their eggs. Intense itching at the anus, especially at night, is the most constant symptom. The disturbances described under common round-worm infection may also occur.

Treatment.—Copious injections of the infusion of quassia, lime-water, or of salt and water (1 dram to 1 pint) are useful in removing the females from the rectum. To rid the small intestine of the young parasites santonin and calomel should be given by the mouth. Care should be taken to prevent auto-infection.

Uncinaria Americana and Uncinaria Duodenalis (*Ankylostomum Duodenale*).—These are hookworms of the genus *Uncinaria*, measuring from 8 to 18 mm. in length. The former prevails in the Americas (southern part of the United States, West Indies, South America), and the latter in Europe, Asia, and Africa. Their habitat is the small intestine.

Uncinariasis, or ankylostomiasis, is a common disease in tropical and subtropical countries. In temperate regions it prevails chiefly among miners. The eggs of the parasite escaping with the feces bring forth embryos which in a few days develop into larvæ. The latter may enter the digestive tract directly through food or water, but it has been shown that in the majority of cases entrance to the bowel is effected indirectly by way of the skin, the route being from the blood to the heart, from the heart to the lungs, from the lungs to the larynx, and thence to the esophagus, stomach, and bowel. The entrance of the larvæ through the skin, usually that of the feet or legs, is followed by an eruption known as ground itch.

Symptoms.—Anemia, more or less severe, is the chief symptom. Eosinophilia is usually present. Digestive disturbances and mental lassitude are frequently observed. The diagnosis depends upon the detection of the eggs or the adult worms in the stools.

Treatment.—Thymol, in doses of 30 grains, repeated in two hours, is the best remedy. A saline purge should be given before and after the anthelmintic, and solvents of thymol, like oil and alcohol, should be avoided on the day of treatment.

Trichina Spiralis.—This is a minute worm derived from the hog. Man is infected by eating raw or rare pork containing encysted larvæ. The latter pass into the small intestine, where they develop into adult worms. The females penetrate the lymphatic spaces, and there bring forth young, which are carried by the lymph or blood to the muscles, where they develop into encysted larvæ. Trichinous capsules, impregnated with lime, are visible to the naked eye. The hog acquires trichiniasis by eating scraps of infected pork or infected rats.

Symptoms of Trichiniasis (Trichinosis).—No decided symptoms develop unless the infection is severe. In well-marked cases gastro-intestinal disturbances appear on the second or third day. These disturbances consist in colicky pains, nausea, vomiting, and serous diarrhea.

In from one to two weeks symptoms of an acute myositis develop. These include severe muscular pain and soreness, edema, especially of the face, profuse sweating, and remittent or intermittent fever. Hoarseness from involvement of the larynx and dyspnea from involvement of the diaphragm are often noted. Eosinophilia is almost constantly present. Delirium and stupor are not uncommon in severe infections. At times the symptoms closely resemble those of typhoid fever. In favorable cases recovery ensues in from two to eight weeks.

Diagnosis.—*Typhoid Fever.*—The history, the presence of eosinophilia, of intense muscular soreness, of edema, of parasites in the stools or in a fragment of muscle removed from the arm, and the absence of a typical rash and of the Widal reaction will lead to a correct diagnosis.

Prognosis.—This depends upon the number of worms ingested. Early diarrhea is favorable. The mortality ranges from 5 to 30 per cent.

Treatment.—The most efficient prophylactic measure is the thorough cooking of all pork products. In the first stage cathartics are indicated. Anthelmintics—santonin and thymol—have been recommended. After migration, the indications are to relieve pain by means of opiates, hot baths, and warm embrocations, and to support the strength by concentrated liquid diet and stimulants.

Filaria Bancrofti (Filaria Sanguinis Hominis).—This is a small, thread-like worm, chiefly met with in warm climates. The adult occupies the lymphatics, and the female brings forth actively motile larvæ, which find their way into the circulating blood. It is a curious fact that the larvæ are found in the blood chiefly at night. The medium of infection is the mosquito, in the body of which the larvæ reach a certain stage of development before being again transmitted to man.

Symptoms.—These are not always present, but chyluria, lymph scrotum, lymph varices, and elephantiasis are not uncommon manifestations.

Treatment.—Thymol and methylene-blue have been recommended, but they are of doubtful utility.

Filaria Loa (Filaria Diurna).—This is a common parasite on the west coast of Africa. The worm occupies the connective tissues throughout the body and often approaches the surface near the eye, causing an edematous swelling with itching, and, at times, more or less intense conjunctivitis. The treatment consists in removing the parasite under local anesthesia.

DISORDERS OF METABOLISM

GOUT

(Podagra)

Definition.—A disturbance of metabolism, characterized in its typical form by deposits of sodium biurate in the joints and other structures, and by recurrent attacks of arthritis.

Etiology.—Gout most frequently develops in the fourth and fifth decades. It is more common in males than in females. It is often hereditary. The excessive use of wines or malt liquors, overeating, sedentary habits, nervous strain, and chronic lead-poisoning are predisposing factors.

Pathology.—The pathology of gout is still obscure. It is generally conceded that the disease is in some way associated with excess of uric acid compounds in the blood; but whether these compounds are the sole cause of the constitutional disturbances, and whether the excess in the blood is due to increased formation or diminished excretion, or both, are questions that await solution.

The only distinctive anatomic lesions of gout are those of the joints. These consist of deposits of sodium biurate (tophi) in the cartilages and fibrous tissues and secondary inflammatory changes. In long-continued cases the joints become irregularly enlarged and stiff. Ultimately ulceration of the superficial tissues may ensue, with the discharge of the uratic concretion. The small joints of the feet and hands are usually the first to be affected, but subsequently other joints, like those of the ankles, wrists, and elbows, become involved. Uratic deposits are often found also along the tendons, in the external ear, in the nose, and in various other parts.

SYMPTOMS.—**Acute Go**
ceded by certain prodrom
ness, irritability, dyspepsia
cretion becoming scanty, hi
The arthritic phenomena
early morning hours, and
swelling in the ball of the
so tender that the slightes
a reddish-purple color; its s
veins are full and distinct.
perature is moderately elev
quickened. Toward dayligh
tent and the patient falls as
paratively comfortable, but
several successive nights.
apart, but as they multiply t
the patient is seldom entirel

Retrocedent Gout.—This
which the arthritic phenom
gastric, cardiac, or cerebral

Chronic Gout.—The join
become stiff, irregularly e

with in America than the articular variety. It presents the following clinical features:

Gastro-intestinal Phenomena.—The tongue is usually coated and the breath heavy; the appetite is variable, sometimes it is lost, at other times it is inordinate; acid eructations, heartburn, and flatulence are frequent gastric symptoms.

Urinary Phenomena.—The urine is scanty, high-colored, of high specific gravity (1025 to 1035), and on standing throws down an abundant brick-dust sediment. The solids render the urine irritating, so that dull aching in the loins and burning in the penis after micturition are common symptoms. A trace of sugar is sometimes detected on chemical examination. The urine often stains the clothes red.

Circulatory Phenomena.—These consist in increased arterial tension, accentuation of the second aortic sound, and a tendency to arteriosclerosis.

Nervous Phenomena.—These are extremely varied, and include headache, vertigo, disturbed sleep, tinnitus aurium, depression of spirits, impairment of memory, loss of energy, irritability, and neuralgic pain in various parts of the body.

Complications and Sequelæ.—These include: Chronic interstitial nephritis, arteriosclerosis, hypertrophy of the heart, angina pectoris, apoplexy, chronic bronchitis, and certain cutaneous affections—chronic eczema, urticaria, and psoriasis.

Diagnosis.—*Acute Rheumatism.*—This more commonly affects the larger joints; it is markedly migratory; it is associated with higher fever and more copious perspiration; and it shows far greater tendency to endocardial and pericardial inflammations.

Rheumatoid Arthritis.—The family history and habits of the patient, the involvement of joints rarely attacked in gout (temporomaxillary, thumb, cervical vertebræ, sternoclavicular), the peculiar deformities, the special joint changes (atrophy or new formation of bone), the absence of tophi, and the muscular atrophy will serve to distinguish rheumatoid arthritis from gout.

toms subside. Alkalis are
of water should be encour
lieved by a full dose of bl
or phenacetin may be requi
affected part should be e
wool, or covered with wa
soaked in lead-water and lau
and non-stimulating.

Chronic Gout.—As regard
are of the utmost importa
composed for the most part
lent vegetables and eggs, is
likely to disagree are veal,
quettes, concentrated soups,
and beans) pastry, sweets,
wines. Some patients are exc

Water-drinking between
more should be eaten than
hunger. The patient shoul
avoid as far as possible e
changes. Systematic exer
beneficial. If active

sional use of calomel or blue-mass at night, with a saline in the morning, is often of value. Among the special remedies advocated for gout may be mentioned alkalis and alkaline mineral waters, colchicum, guaiac, arsenic, and iodids.

Of these, the alkalis, especially the vegetable salts of potassium or lithium, are the most useful. Colchicum is most effective in the acute paroxysms, although small doses with alkalis may be of benefit in the interval. Guaiac probably ranks next in efficacy to the alkalis. The prolonged use of arsenic in small doses seems to be of some value. Iodids are sometimes of service in relieving the concomitants and sequels of gout, but have little, if any, effect upon the disease itself. Salicylates relieve pain, but are distinctly inferior to colchicum.

Chronic affections of the joints are best treated by gentle massage, friction, and warm sulphur baths.

RICKETS

(Rachitis)

Definition.—A constitutional disease of early childhood, characterized chiefly by defective nutrition of the osseous structures.

Etiology.—Rickets is most common between the sixth month and the third year of life. Poor nutrition of the mother, bad air, lack of sunlight, and, above all, improper feeding, are important etiological factors, hence the disease is observed most frequently in large cities and among the children of the poor.

Pathology.—The changes indicate tardy bone-formation with undue preparation for it. They consist of marked increase of cartilage at the ends of the bone, excessive production of osteoid tissue by the medullary and periosteal structures, irregular and deficient calcification, and resorption of bone from within out of proportion to that formed by the periosteum. In consequence of these defects the bones are softened, irregularly enlarged, especially in the region of the epiphyses, and often bent or otherwise distorted. With the arrest of the dis-

perspiration, especially abou
disinclination to move or to
peresthesia; delayed or irre
disturbances; pallor, emacia
muscles, and undue protubera
owing to muscular atony, fl
liver and spleen.

Skeletal Features.—The he
more or less square; the font
normal and the sutures are
parietal bones may be abnor
tabes); enlargements or beads
of the osseous and cartilagino
rosary), the sides of the chest
thrust forward (pigeon-breast
son's groove) is sometimes s
belly; curvatures of the spin
enlargements are observed in
leg bones with the production
common; finally, the pelvic out

Complications.—Green-stick
convulsions, involuntary mict
stridulus sometimes

and fresh meat juice are sometimes useful additions. Cod-liver oil, phosphorus (5 to 15 minims of the elixir), iron and arsenic are serviceable remedies.

℞. Olei morrhuæ	f℥iss
Olei gaultheriæ	
Pulveris acaciæ	āā q.s.
Syrupi ferri iodidi	fʒii–fʒiii
Aquæ	q.s. ad f℥iii.—M.

SIG.—A teaspoonful to a dessertspoonful after meals.

DIABETES MELLITUS

Definition.—A disorder of metabolism characterized by impairment of the ability of the tissues to utilize carbohydrate, and manifested by persistent hyperglycemia, glycosuria, and ultimately acid intoxication.

Etiology.—The disease is most common between the ages of thirty and sixty. Hebrews appear to be especially prone to it. Males are more frequently affected than females. Overeating and sedentary habits favor its occurrence, and therefore it is much more prevalent among the well-to-do than among the poor. Hereditary influence may be traced in some cases. Among determining causes may be mentioned mental strain, acute infections, and injury of the brain or spinal cord.

Pathology.—The underlying cause of diabetes is still undetermined. Lesions of the pancreas involving the islands of Langerhans, however, are found in the vast majority of cases, and it is probable that failure of an internal secretion of the pancreas is the most important etiologic factor. Possibly other glands with which the pancreas is in relation—adrenal, hypophysis, and thyroid—may at times be concerned in the process. The effect of lesions of the central nervous system, such as puncture of the floor of the fourth ventricle, is not well understood. It may be that nervous influences affect the pancreatic function or result in imperfect storage or retention of glycogen in the liver. In addition to disease of the pancreas, the liver is usually enlarged, and nephritis is present in about one-half of the fatal cases.

Failure of the tissues to utilize carbohydrate results in an excess of glucose in the blood (hyperglycemia), and to this feature is attributable the glycosuria, thirst, polyuria, emaciation, and many other symptoms of the disease. A secondary result of the disordered carbohydrate metabolism is the loss of ability to oxidize completely the fats, leading to the appearance of acetone bodies in the urine (ketonuria), first acetone, then diacetic acid, and later in severe cases, betaoxybutyric acid.

Symptoms.—*General Phenomena.*—The onset is usually insidious, but it may be abrupt. As a rule, the first symptoms to attract attention are weakness, excessive thirst, frequent micturition, and polyuria. Pruritus, especially of the genitalia, may be an early manifestation. The appetite may or may not be inordinate. In severe cases emaciation is marked. The skin is dry and harsh; the saliva is scanty; the tongue is frequently red and glazed; the teeth readily decay; and the bowels are usually constipated. Impotence is common; the knee-jerks are diminished or lost, and many patients complain of neuralgic pains and muscular cramps. The most important nervous phenomenon, however, is coma, which is the direct cause of the fatal issue in more than one-half of the cases. It is due to acidosis (β-oxybutyric acid), and frequently develops as the result of overexertion, intercurrent disease, or nervous shock, but it may occur as the last event without any definite exciting cause. It is often preceded by headache and somnolence, and is accompanied by air-hunger (long-drawn inspirations), a fruity odor to the breath, digestive disturbances, and symptoms of collapse. It is almost always fatal in from a few hours to a few days.

The Urine.—The urine is increased in amount, the daily output varying from 3 or 4 liters to 10 liters or more; it is pale in color, of increased specific gravity (1025–1040), and contains sugar, and frequently one, two or all three of the acetone bodies. The sugar content varies from 0.5 to 10 per cent., and the daily output from 50 to 1000 grams. Albumin and casts are also present in many cases. The total nitrogen and ammonia are increased, the latter often to 8 grams or more (normally from 0.5 gram to 1.5 grams). The excess of alkali is required for the neutralization of the organic acids and the amount of ammonia excreted is therefore an approximate measure of the degree of acidosis, 2 grams corresponding to about 6 grams of β-oxybutyric acid and 5 grams to about 20 grams of β-oxybutyric acid. The amount of urine and the sugar and acid contents usually diminish markedly before the onset of coma.

The Blood.—Hyperglycemia is a constant feature, the concentration of sugar often rising to 0.3 or 0.5 per cent. (normally 0.06 to 0.1 per cent.). As the amount of sugar excreted depends to a great extent upon the degree of renal permeability, estimation of the blood-sugar provides a much more reliable criterion of the severity of the disease than estimation of the urinary sugar.

Complications.—The most common complications result from diminished resistance to bacterial infection and comprise boils, carbuncles, tuberculosis, and pneumonia. The last is not rarely followed by gangrene of the lung. Skin eruptions, especially eczema, are frequently observed. Nephritis occurs in at least one-half of the cases. Peripheral neuritis is common. Defective vision from cataract, retinitis or optic neuritis may occur. Gangrene of the leg (obliterating endarteritis) is not uncommon in elderly subjects.

Diagnosis.—Recognition of the disease in its early stages is only possible through routine examination of the urine. Transient glycosurias and other forms of mellituria (lactosuria, pentosuria, etc.), must be excluded. In alimentary glycosuria the administration of 100 grams of glucose two hours after a light breakfast may be followed by the appearance of sugar in the urine, but the whole amount is less than 2 grams and the excretion never persists more than six hours. Glycosuria on a diet containing starch but no sugar should be regarded as prima facie evidence of diabetes.

Prognosis.—This depends upon the severity of the disease and the faithfulness with which appropriate treatment is carried out. The outlook is, as a rule, more favorable in obese, middle-aged persons than in thin, young subjects. In children the disease is quickly fatal. Generally speaking, those are mild cases in which 3 ounces of bread (about 60 grams of starch) can be metabolized without the occurrence of glycosuria; those are moderately severe cases in which glycosuria occurs with this amount of carbohydrate but disappears with a strict meat-fat diet; and those are severe cases in which glycosuria still persists with a carbohydrate-free diet. The duration of the disease varies from several weeks in the very acute cases to twenty years or more in the very chronic ones.

Treatment.—This is chiefly *dietetic*. The indications are to maintain nutrition, to increase the tolerance for carbohydrates, to lessen hyperglycemia, and to prevent or diminish acidosis. In mild cases (except in childhood and adolescence) the diet may contain the full amount of carbohydrate that can be tolerated without causing glycosuria, but a week of strict (carbohydrate-free)[1] diet should be interposed every four or five weeks. In severe cases with acidosis, the diet should have a low nitrogen content and contain the carbohydrate best tolerated (oatmeal, potato, fruit) in such quantity that the glycosuria is kept at a minimum and the body weight is not reduced. Frequent periods of strict diet must be introduced, however, to lessen the hyperglycemia, and during these periods from ½ to 1 ounce of sodium bicarbonate should be given daily to control the acidosis. In cases requiring rigorous treatment alcohol in moderate amounts, in the form of whiskey, brandy, or white wine, is useful in supplying additional energy and aiding in the digestion of fats.

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[1] **Articles Permitted**.—*Animal Foods.*—Meats of all kinds (except liver), fish (except oysters and clams), eggs, and soups without flour.

Fats.—Butter, lard, olive oil, cheeses of all kinds, and cream (small amounts).

Vegetables.—Celery, lettuce, cucumbers, cresses, asparagus, egg-plant, spinach, string beans, tomatoes, cauliflower, cabbage, squash, mushrooms and pickles.

Desserts.—Custards and ice-cream containing no starch and sweetened with saccharin.

Beverages.—Tea, coffee or lemonade sweetened with saccharin, mineral waters, whiskey, brandy, Rhine wine or Burgundy.

Articles Prohibited in Strict Diet.—Bread, cake, biscuit, and pastry of all kinds (most gluten breads contain a considerable amount of starch and are indigestible); fruits of all kinds; cereals of all kinds; macaroni, potatoes, carrots, beans, beets, turnips, corn, peas, and parsnips; milk, cocoa, and chocolate; malt liquors, cider, sweet wines, and liqueurs; preserves and ice-cream.

General hygienic measures are of great importance, especially the prevention of mental and physical overexertion, worry, and excitement. A moderate amount of regulated exercise is beneficial in mild cases, but considerable rest is absolutely necessary in severe forms of the disease.

Drugs.—Tonics, like arsenic, iron and strychnine are often useful. Opium proves efficacious in some cases. It is best given in the form of codein (½ grain three or four times a day). Salicylates have been strongly recommended. Bromids are serviceable in subduing nervous manifestations. Alkaline carbonates and alkaline mineral waters have long enjoyed a reputation.

Upon the recognition of the early signs of coma, a moderate amount of readily digestible carbohydrate should be added to the diet. Absolute rest should be enforced, saline laxatives and diuretics (theobromin, caffein), should be administered, and large doses (1 to 2 ounces) of sodium bicarbonate should be given daily. Developed coma is rarely relieved by *intravenous* injections of 4 per cent. solution of sodium carbonate (a liter, if possible, and repeated if necessary at end of six hours).

DIABETES INSIPIDUS

Definition.—A chronic condition, characterized by the excretion of large quantities of pale, limpid urine of low specific gravity and free from albumin and sugar.

Etiology.—The disease is most common between the ages of twenty and thirty. More males are affected than females. Heredity may be an important factor. In some cases polyuria is the primary symptom and is associated with a lesion at the base of the brain (probably involving the pituitary gland). Frequently the lesion is of syphilitic origin. In other cases the kidneys seem to be incapable of secreting a concentrated urine, that is, urine containing a normal percentage of salt and urea. In a third group of cases there is apparently a primary polydipsia, the result of a neurosis.

Symptoms.—The onset may be gradual or sudden. The chief symptom is the passage of large quantities (5 to 20 liters) of sugar-free urine, of low specific gravity (1005–1001), over a long period. Accompanying the polyuria there is usually insatiable thirst. The skin and mouth are dry; the bowels are usually constipated, and headache, lumbar pains, and nervous irritability are common. In many cases there is neither weakness nor emaciation. Complications are rare.

Diagnosis.—The high specific gravity of the urine and the presence of sugar will serve to distinguish *diabetes mellitus* from diabetes insipidus.

Chronic interstitial nephritis may be recognized by the presence of tube-casts in the urine, the albuminuria, and cardiovascular signs.

Prognosis.—The disease usually continues for years and is not often directly fatal. Complete cure is rarely effected by treatment, even in syphilitic cases, but it occasionally occurs spontaneously.

Treatment.—No benefit is derived from restricting the quantity of water, except perhaps, in the neurotic cases. In the renal type restriction of the salt and protein intake is sometimes of service. Considerable alleviation of symptoms often follows the use of antiluetic remedies in syphilitic cases. Other remedies sometimes of value are valerian (3 to 5 grains of zinc valerate, thrice daily), ergot (10 minims the fluid extract thrice daily), bromids, strychnin, and lladonna.

SCURVY

(Scorbutus)

Definition.—A disease characterized by marked weakness, iemia, hemorrhages from the mucous membranes and into ie skin, and a tendency to a spongy state of the gums.

Etiology.—In adults the chief etiologic factor is a lack of esh food, especially of fresh vegetables and meat. Unhygienic irroundings, exposure to cold and wet, and mental depres- on are cited as predisposing causes. The pathogenesis of the isease is still undetermined.

Symptoms.—These include anemia with great weakness nd lassitude; spongy, bleeding gums with fetor of the breath nd loosening of the teeth; subcutaneous ecchymoses and emorrhages from the mucous membranes; and brawny in- uration of the muscles in various parts of the body from a inguineous transudation.

An infantile form of scurvy (*Barlow's disease*) sometimes llows the prolonged use of condensed milk, sterilized milk, proprietary foods. The characteristic symptoms are: allor, tenderness or pain in the legs or back on handling, ight swelling, especially about the diaphyses, immobility of ie legs (pseudoparalysis), ecchymoses, and hematuria. The ums are not affected unless the child has teeth.

Prognosis.—Recovery usually occurs rapidly under ap- ropriate treatment, but neglected cases are often fatal.

Treatment.—The diet should include fresh vegetables—otatoes, lettuce, cabbage, and onions—with several ounces lemon-juice daily. Iron is of service. The mouth should e cleansed at frequent intervals with some antiseptic ash.

In infantile scurvy good results follow the use of fresh milk, eef-juice, and orange-juice.

BERIBERI

(Endemic Multiple Neuritis; Kakke)

Definition.—A form of polyneuritis occurring endemically in tropical and subtropical countries and characterized by disturbances of sensation, of motion, and of the circulation.

Etiology.—Beriberi is especially prevalent in Japan, China, the Philippine Islands, India, and parts of South America. Although the etiology of the disease has not been definitely determined, the experiments of Fraser and Stanton, confirmed by other workers, are conclusive to the effect that a defective diet is an essential factor. Outbreaks have frequently been associated with a diet of rice freed from certain substances contained in the pericarp (polished rice). Overcrowding, bad hygienic surroundings, and exposure to wet are predisposing factors. Young adults are chiefly affected.

Symptoms.—To the usual symptoms of polyneuritis (paresis, especially in the lower extremities, pains, paresthesia, hypesthesia, tenderness of the nerve-trunks, loss of deep reflexes, diminished electric excitability, and muscular atrophy) are added various disturbances of the circulation, such as palpitation, dyspnea, weakness of the pulse, and signs of venous congestion. In many cases more or less edema of the feet and legs is also noted.

Varieties.—*Acute pernicious* forms occur in which the onset is rapid, grave disturbances of the circulation soon develop, and death ensues in the course of a few days from heart failure and edema of the lungs. So-called *wet* and *dry* forms are also observed. In the former there is marked edema and often effusions into the serous sacs. In the latter there is little or no edema, but muscular atrophy is very pronounced. Finally, in all outbreaks there are many *rudimentary* cases in which the only symptoms are weakness in the legs, paresthesia, and palpitation.

Prognosis.—The mortality has varied in different outbreaks from 2 to 60 per cent. Generally speaking, the prognosis is good, except in pernicious cases, when the patient can be placed under proper hygienic conditions. The average duration of cases of moderate severity is between three and eight weeks.

Treatment.—Avoidance of overcrowding and the use of a diet rich in nitrogneous food are the most important prophylatic measures. A person sick with beriberi should be confined to bed and placed on a light and nutritious diet. Saline laxatives appear to be useful in the early stages. Morphin is serviceable when there is much pain or dyspnea. Caffein and the vegetable salts of potassium are worthy of trial if there is marked edema. Venous congestion is sometimes favorably influenced by venesection. Digitalis is of doubtful value. In the chronic stages the treatment is that laid down for multiple neuritis (see p. 496).

DISEASES OF THE NERVOUS SYSTEM

DISTURBANCES OF MOTION

THESE consist, for the most part, of loss of power or paralysis, and manifestations of motor excitation, such as convulsions, choreiform movements, and tremors.

Paralysis.—The paralysis may be irregularly distributed, or it may involve a single member, when it is termed *monoplegia;* a lateral half of the body, when it is termed *hemiplegia;* or the body from the waist down, when it is termed *paraplegia.*

Irregular paralysis may result from:

1. Disseminated lesions in the central nervous system, such as occur in multiple sclerosis and cerebrospinal syphilis.
2. Lesions in the basal ganglia—pons, crura cerebri, medulla—when it is often associated with headache, vomiting, vertigo, and optic neuritis.
3. Acute poliomyelitis. This occurs chiefly in children. The paralysis develops rapidly, often within a few hours; it tends toward recovery in some parts, and to permanent disability and atrophy in others.
4. Progressive spinal muscular atrophy or chronic poliomyelitis. This occurs in middle life. The paralysis usually begins in the small muscles of the hand; is accompanied by atrophy; and very gradually spreads until a large part of the body is affected.
5. Muscular dystrophies. These commonly develop during childhood; involve the muscles of the calves, trunk, face, or shoulder-girdle; are associated with progressive atrophy; and are usually traceable to heredity.

6. Multiple neuritis. This is recognized by the history, pain, disturbances of sensation, loss of tendon reflexes, muscular atrophy, and tenderness over the nerve-trunks.

7. Syringomyelia. This is rare. It usually develops in early adult life, and may be recognized by the progressive atrophy of the muscles, spinal curvature, various trophic disturbances, and loss of thermic and painful sensibility, with preservation of tactile sensibility.

Monoplegia may result from:

1. A focal lesion in the cortical area of the brain. This may be recognized by the history, increased tonicity of the muscles, exaggerated reflexes, and the absence of atrophy, of sensory disturbances, and of changes in electrical excitability.

2. A lesion of the peripheral nerves. This may be recognized by the history, loss of reflexes, loss of muscular tone and later, atrophy, changes in electrical excitability, and, perhaps, the presence of sensory disturbances.

3. Hysteria. This may be recognized by the history, the emotional instability and pathologic suggestibility of the patient, and the absence of wasting, alterations in tendon reflexes, and all other signs of organic disease.

Hemiplegia may result from:

1. A diffuse lesion of the motor cortex. The paralysis is on the opposite side of the body and is unassociated with anesthesia.

2. A lesion of the internal capsule. This is the most common seat of hemorrhage; the paralysis is on the opposite side of the body and is rarely associated with anesthesia.

3. A lesion of the crus cerebri. This frequently produces hemiplegia and hemianesthesia on the opposite side, and paralysis of the oculomotor nerve on the side of the lesion, indicated by dilated pupil, strabismus, and ptosis.

4. A lesion of the pons. This frequently produces hemiplegia and hemianesthesia on the opposite side, and facial paralysis on the side of the lesion.

7. A unilateral lesion high in the cord (very rare). This

Paraplegia may result from:

1. Acute myelitis. This dev
days, usually as the result of
infection; the paralysis is flacci
comes spastic; sphincter disorde
a "girdle sense" are present, an
to bed-sores and cystitis.

2. Landry's disease (acute as
sembles myelitis in mode of on
associated with sphincter disord
other trophic disturbances.

3. Acute poliomyelitis. This
it comes on suddenly, and is ush
quently, with pains in the liml
rapidly, affects groups of muscles
is flaccid and atrophic, and ten
parts; sphincteric incontinence
and there is no tendency to bed-s

4. Compression of the cord (
aneurysm, etc.). The suggestiv
tenderness, rigidity, and deforn
limbs; paresthesia, followed by a

6. Fracture or dislocation of the spine. The history of injury, the local deformity, the abrupt onset of flaccid paraplegia, with anesthesia and sphincteric interference, and the marked tendency to bed-sores and cystitis are the diagnostic features.

7. Some forms of multiple neuritis. The history of some antecedent infection or intoxication, diffuse pain, tenderness of the nerve-trunks and calves, the special incidence of the palsy and sensory disturbances on the peripheral parts of the limbs, muscular atrophy, and loss of tendon reflexes, without sphincter disorder, girdle sensation, or any marked tendency to bed-sores, will point to neuritis.

8. Amyotrophic lateral sclerosis and lateral sclerosis. Both of these conditions develop gradually, and are marked by motor weakness, spasticity of the muscles, and exaggerated reflexes, without sensory symptoms. In the former there is also muscular atrophy.

9. Disseminated sclerosis. In this there is spastic paresis in association with intention tremor, scanning speech, nystagmus, and visual defects.

10. Cerebral palsies of childhood. These are characterized by spastic paralysis, mental deficiency, stunted physical development, and, also, in many cases, by choreiform movements and epileptic seizures.

11. Caisson disease (divers' paralysis). The history will establish the diagnosis.

12. Hysteria. This may be recognized by the emotional instability and suggestibility of the patient, and by the absence of wasting, sphincteric disturbances, alterations in deep reflexes, and all other signs of organic disease.

Convulsions.—Convulsions are involuntary muscular contractions, interrupted or long-continued, resulting from excessive irritation or undue irritability of the motor centers.

Interrupted contractions are termed *clonic;* long-continued contractions, *tonic.*

Convulsions may be general or local. The term *spasm* is sometimes applied to the latter.

General clonic convulsions may be due to:

1. Idiopathic epilepsy. This condition usually develops before the age of twenty-five, and the convulsions are general and are unassociated with any definite cause.

2. Organic brain disease. In this condition there may be a history of syphilis or of injury; the convulsions may be local, or begin as such and become general; and there may be concomitant symptoms of cerebral disease.

3. Various intoxications. Alcoholism, the infectious fevers, and uremia are frequently associated with convulsions.

4. Reflex irritation. Such convulsions are usually observed in young children, and result from gastric irritation, an adherent prepuce, intestinal parasites, or teething.

5. Cerebral anemia. Such convulsions are sometimes seen after profuse hemorrhage, and in degeneration of the heart muscle (Adams-Stokes syndrome).

Tonic convulsions may result from:

1. Tetanus. This may be recognized by the presence of a wound, the early involvement of the muscles of mastication, and the persistent rigidity of the muscles of the back.

2. Cerebrospinal meningitis. This may be recognized by the headache, fever, rigidity of the neck, Kernig's sign, local palsies, and turbidity of the fluid obtained by lumbar puncture.

3. Strychnin-poisoning. This may be recognized by the history, the intermittent character of the convulsions, the absence of fever, and the escape of the muscles of the jaw until very late.

4. Tetany. In this condition the extremities are chiefly involved; the contractions are tonic and intermittent, involve bilaterally symmetrical groups of muscles, and can be reproduced by pressure on the nerves of the affected member.

Hysteric Convulsions.—The convulsions of hysteria may simulate those of epilepsy, but, unlike the latter, they are rarely attended by complete loss of consciousness, pupillary rigidity, biting the tongue, or involuntary passage of urine. Moreover, in hysteria the movements are usually tonic, or if clonic, appear purposive, and the paroxysms are of long duration.

Eclampsia.—This term is applied to a sudden attack of convulsions, the result of a temporary cause, such as the convulsions of childhood resulting from reflex irritation, and the convulsions of pregnancy resulting from toxic materials retained in the blood.

Local Convulsions or Spasms.—*Facial spasm* may result from—(1) Irritative lesions at any point in the course of the seventh nerve from the cortex to the periphery; (2) peripheral facial palsy (tonic spasm); (3) hysteria; (4) trigeminal neuralgia (*tic douloureux*); (5) simple tic, in which there are natural but purposeless contractions of psychogenic origin; (6) tic convulsif, in which there are abrupt contractions of the facial muscles and other muscles, with explosive utterances and a tendency to use obscene words and to repeat words and movements made by other persons.

Transitory spasms of one arm or one leg are usually manifestations of Jacksonian epilepsy (focal epilepsy), but they sometimes result from hysteria.

Spasm of the hand developing when the member is put to use may result from writers' cramp, myotonia congenita (Thomsen's disease), or hysteria.

Spasm of the cervical muscles (wry-neck, torticollis) may be due to: (1) Exposure to cold (acute "stiffneck"); (2) congenital shortening of sternomastoid muscle; (3) irritation of the spinal accessory nerve caused by disease of the vertebræ or meninges in the upper cervical region; (4) obscure functional disturbance of the cortical centers (mental torticollis, psychogenous tic).

Spasms of the larynx, esophagus, and diaphragm (hiccup) have already been discussed.

Choreiform Movements.—These are involuntary, jerky, quickly changing muscular contractions, resulting in movements of a purposeless nature. They may result from:

1. Infectious chorea (St. Vitus' dance). This disease occurs chiefly in children, usually lasts from six to ten weeks, is prone to recur, and is frequently complicated by endocarditis. A severe form occurring chiefly in women during pregnancy and characterized by violent movements, fever and delirium, is known as chorea insaniens.

2. Huntingdon's chorea. This affection occurs in adult life and is hereditary. The movements in time become general involving the muscles of speech and deglutition, and are associated with a progressive mental deterioration.

3. Organic brain disease. Choreiform movements are frequently observed in the cerebral palsies of children, and occasionally they occur in adults on one side of the body before an attack of apoplexy (prehemiplegic chorea, or in the paralyzed members after an attack (posthemiplegic chorea).

4. Habit chorea (tic). This disorder occurs in persons of a nervous temperament, sometimes as the result of the frequent repetition of volitional acts (mimicry). The movements which are most commonly facial are purposeful, partially or completely under the will, and confined to a single muscle or group of muscles.

5. Senile chorea. Occasionally old persons with arteriosclerosis and degenerative processes in the cerebral cortex become subject to chorea.

6. Hysteria (Henoch's electric chorea). In this condition the movements are sudden and rhythmical like those caused by electric shocks and are associated with other stigmata of hysteria.

7. Friedreich's ataxia. This is a family disease, developing in early life, and characterized by ataxia, choreiform movements, a loss of deep reflexes, nystagmus, defective speech, spinal curvature and club-foot.

8. Paramyoclonus multiplex. A very rare disease, of

unknown origin, characterized by violent clonic spasms of the muscles of the trunk and upper part of the limbs. The spasms are bilateral and rarely, if ever, involve the forearms, hands, legs, or feet. They occur only at intervals and can usually be brought on by irritation of the skin or tendons, or by excitement.

Athetosis.—This term was suggested by Hammond to designate certain movements occurring chiefly in the hands and feet, and characterized by slow twisting, intertwining, separation, and extension of the fingers and toes. Athetosis is observed chiefly in cerebral hemiplegia, especially the infantile form, and in lesions of the optic thalamus.

Tremors.—A tremor is a rhythmic vibratory movement due to the alternate contraction and relaxation of antagonistic muscles. Tremors are observed in the following conditions:

1. They may exist from birth unassociated with other symptoms.
2. They may depend upon a lowered tone of the nervous system, being frequently observed in exophthalmic goiter, neurasthenia, and in the convalescence from acute disease.
3. They may be toxic, resulting from alcoholism, morphinism, mercurial poisoning, etc.
4. They may be due to old age.
5. They are frequently a symptom of organic disease of the brain and cord; as such, they are met with in paretic dementia, and especially in disseminated sclerosis. In the latter they arise only during purposeful movements (intention tremors).
6. They constitute an important symptom in paralysis agitans.
7. They may be hysterical.

The Gait.—*The Ataxic Gait.*—In locomotor ataxia the patient raises the foot high, throws it forward, and brings it down suddenly, so that the whole sole comes in contact with the floor at once.

Spastic Gait.—In spastic paraplegia the movements are stiff, the knees come together, the leg drags behind, and the toe catches the ground.

Festination.—This term is applied to the gait of advanced paralysis agitans; in walking, the body inclines more and more forward, and the steps grow faster and faster until the patient falls, straightens himself by a supreme effort, or finds support in some neighboring object.

Steppage Gait.—In chronic multiple neuritis the patient raises the foot high, turns the toe up, and brings the heel down first.

The Gait of Pseudomuscular Hypertrophy.—The feet are wide apart, the belly protrudes, and the movements are clumsy and waddling.

Titubation.—This term is applied to the peculiar gait observed in lesions of the cerebellum. It resembles the gait of locomotor ataxia, but is much more staggering, the body swaying like that of a person intoxicated. With the ataxia there is a marked vertigo, which usually disappears when the patient lies down.

The Reflexes.—**The Knee-jerk, or Patellar Tendon Reflex.**—This is obtained by tapping the quadriceps tendon between its insertion and the patella while the leg is crossed over its fellow.

The knee-jerk is increased in the following conditions:

1. In cerebral lesions involving the pyramidal tracts (diminution of inhibition), as in hemiplegia from apoplexy, tumor, etc.
2. In compression or partial destruction of the cord above the lumbar region, as in Pott's disease or tumor of the cord.
3. In disseminated cerebrospinal sclerosis, lateral sclerosis, and amyotrophic lateral sclerosis.
4. In irritability of the cord, as in mania, hysteria, strychnin-poisoning, and spinal meningitis.

The knee-jerk is diminished or absent in the following conditions:

1. In the various forms of primary muscular atrophy.

2. In lesions of the nerves which cut off the impulse from the cord—as neuritis.

3. In lesions of the posterior columns of the cord, as in locomotor ataxia.

4. In poliomyelitis, both acute and chronic.

5. In transverse lesions of the cord completely destroying the reflex arc, as in lumbar myelitis, fracture of the vertebræ, etc.

6. In exhaustion of the spinal centers, as after prolonged muscular exertion.

Ankle-clonus.—This consists of vibratory movements of the foot, obtained by supporting the *tendo Achillis* with one hand while the foot is strongly flexed with the other. It occurs with lesions that incompletely interrupt the conductivity of the pyramidal tracts, such as primary lateral sclerosis, amyotrophic lateral sclerosis, disseminated sclerosis, degeneration of the lateral columns following cerebral hemorrhage, and compression myelitis.

Arm-jerk.—This is obtained by striking the biceps tendon at the elbow, or the triceps tendon above the olecranon.

Jaw-jerk.—This is obtained by tapping the jaw while the mouth is partially open.

Babinski's Reflex.—This consists in extension of the great toe instead of flexion when the sole of the foot is stroked. It is often normally present in infants. In adults it is suggestive of some disturbance of the pyramidal tracts—meningitis, tumor, hemorrhage, amyotrophic lateral sclerosis, etc.

Kernig's Sign.—This consists in an inability to straighten the leg completely when the patient is in the recumbent posture and the thigh is flexed at a right angle with the pelvis. It is of value in the diagnosis of meningitis.

The Cutaneous or Superficial Reflexes.—These are muscular contractions resulting from irritation of the sensory nerves of the skin. Their nature is imperfectly understood. As they are inconstant in health they are much less serviceable for diagnosis than the deep reflexes. Cutaneous reflexes are frequently lost in those diseases in which the tendon reflexes are exaggerated. The important reflexes of this type are the following:

Abdominal Reflex.—Tickling or shaking the skin of the abdomen causes contraction of the muscles on the side stimulated.

Cremasteric Reflex.—Stroking the inner side of the thigh causes retraction of the testicle.

Plantar Reflex.—Tickling the sole of the foot causes sudden plantar flexion of the toes.

Gluteal Reflex.—Irritation of the skin about the buttock causes contraction of the gluteal muscles.

DISTURBANCES OF SENSATION

These consist chiefly in a loss of sensibility—*anesthesia;* increased sensibility—*hyperesthesia;* subjective sensations, not actually painful—*paresthesia;* and subjective painful sensations—*neuralgias.*

Anesthesia.—Ordinary cutaneous sensibility may be tested by the prick of a pin, by a pinch, or by the faradic current. Diminution of sensibility is known as *hypesthesia.*

Anesthesia or hypesthesia may result from a lesion of (1) the sensory cortex (posterior central convolution); (2) internal capsule; (3) optic thalamus; (4) crus cerebri; (5) pons; (6) medulla; (7) sensory tracts of the cord; (8) peripheral nerves; or from (9) hysteria.

Hemianesthesia.—A loss of sensation in a lateral half of the body. It may result from:

(1) A lesion in the sensory cortex, internal capsule, crus cerebri, pons, or medulla; (2) a unilateral lesion high in the cord (Brown-Séquard's paralysis); or (3) hysteria.

Monanesthesia.—A loss of sensation in one member. It may result from hysteria, from a focal lesion of the sensory area of the cortex, or from a lesion of the nerves supplying the affected member.

Paranesthesia.—A loss of sensation in all parts below the waist. It may result from hysteria, organic diseases of the cord, involving the sensory tracts, or neuritis of the lower extremities.

Analgesia and Thermo-anesthesia.—The former is a loss of sensibility to pain, the latter a loss of sensibility to heat and cold. The association of these two disturbances, together with the preservation of tactile sensation, is known as *dissociative anesthesia.* This phenomenon is most frequently observed in syringomyelia, but it may occur also, though rarely, in spinal tumor, vertebral caries, locomotor ataxia, neuritis, and hysteria.

Astereognosis.—This is an inability to recognize objects by handling them. It usually indicates a lesion of the superior parietal convolution.

The Sense of Space.—The distance at which two points of contact can be recognized as two points. Normally the distance varies in different parts and in different individuals.

On the cheek it is 11–15 millimeters.
On the forehead, 22 millimeters.
On the forearm, 40 millimeters.
On the chest, 45 millimeters.
On the thigh and upper arm, 68 millimeters.
On the leg, 40 millimeters.
On the palm of the hand, 8–12 millimeters.
On the back of the hand, 31 millimeters.

Hyperesthesia is increased insensibility to external impressions. It may be produced by irritation of any portion of the sensory tract.

Paresthesia.—This term is used to indicate certain disagreeable subjective phenomena, such as numbness, tingling, itching, creeping, prickling, etc.

Paresthesia is observed in many conditions, as hysteria, spinal sclerosis, neurasthenia, and injury or inflammation of the nerves. It is frequently observed in elderly persons with arteriosclerosis.

Girdle Sensation.—The sensation of having a girdle or tight band around the trunk. It is frequently observed in locomotor ataxia and myelitis.

Neuralgia.—This consists of paroxysms of severe pain radiating along the line of the nerve-trunks. The pain is relieved by pressure, but there are tender spots (*points douloureux*) where the nerve makes its exit from bony canals or muscular coverings.

Lightning-pains.—This term is applied to the sharp lancinating pains observed in locomotor ataxia. They usually occur in the extremities, and may be mistaken for rheumatism.

Muscle Sense.—This includes the appreciation of the position of the limbs, of active and passive movements, and of pressure and resistance. It is disturbed in locomotor ataxia, and sometimes in brain tumor (parietal region and optic thalamus) and hysteria.

DISTURBANCES OF NUTRITION

These consist chiefly of atrophy of the muscles, changes in the joints, and alteration in the skin and its appendages.

Muscular Atrophy.—Atrophy or wasting of the muscles may result from:

1. Inactivity. Cerebral palsies may thus be associated with gradual wasting (simple atrophy).

2. Certain affections of the muscles themselves, as the various types of progressive muscular dystrophy (simple atrophy).

3. Diseases affecting the anterior horns of gray matter of the spinal cord or the motor nerves (degenerative atrophy).

Changes in Electromuscular Contractility.—A normal response of the muscles to both galvanic and faradic currents usually occurs in *hysteric paralysis* and in *paralysis of cerebral origin.* An increased response to both currents without qualitative change indicates a state of hypersensitiveness of the spinal centers or peripheral nerves, and may be observed in *very recent cases of neuritic paralysis* and in *tetany*. A diminished response to both currents without qualitative change is frequently observed in the *muscular dystrophies*.

Reaction of Degeneration (**DeR**).—This consists in a qualitative change in the electric reaction, a reversal of that occurring in normal muscle. It is obtained only with the *galvanic current* when the electrode is placed over the *muscle*—not its motor nerve or motor point—and occurs in paralyzed muscles which are in certain stages of degeneration owing to a lesion of the ganglion cells in the anterior gray horns of the cord or of the prolongations of these cells in the peripheral nerves. Thus it is observed in *acute* and *chronic poliomyelitis*, in *acute myelitis*, and in *severe forms of neuritis*. In these diseases the affected muscles fail to respond to the faradic current, but still respond to the galvanic current. The responses, however, instead of being prompt and short, as in health, are sluggish and persistent, and, moreover, are reversed in their sequence. Thus, the anodal (positive pole) closing contraction may equal, or at a later period exceed, the cathodal (negative pole) closing contraction, and the cathodal opening contraction may equal or exceed the anodal opening contraction. These reactions may be expressed as follows:

AnClC equals or is greater than CaClC. CaOC equals or is greater than AnOC.

Arthropathies.—An arthropathy is a degenerative affection of the joints, characterized by marked swelling due to effusion, erosion of the cartilages, relaxation and calcification of the ligaments, and atrophy of the heads of the bones. Arthropathies are observed in certain organic diseases of the spinal cord, more especially in locomotor ataxia and syringomyelia.

Ulceration Resulting from Perverted Nutrition.—*Acute Decubitus.*—This term is applied to ulcers appearing in a few hours or days, on parts subjected to pressure, after the occurrence of a severe cerebral or spinal lesion.

Chronic Decubitus.—This term is applied to the ulcers which ultimately appear on parts subjected to pressure in the course of chronic spinal affections.

Perforating Ulcer of the Foot.—This term is applied to an undermining ulcer of the foot most commonly observed in locomotor ataxia. It frequently penetrates the deep structures and involves the bones.

Symmetric Gangrene (Raynaud's Disease).—This is a gangrenous affection involving the fingers, toes, tip of the nose, or ears. It arises spontaneously, and is probably due to a vasomotor spasm.

Trophic Affections of the Skin.—Herpes, scleroderma, vitiligo, chloasma, and "glossy skin" following injuries of the nerve-trunks are illustrations of this class of trophic phenomena.

Trophic Affections of the Hair and Nails.—After injury of the nerves and in neuritis the nails often become dry, brittle, and cracked. Under similar conditions there may be a loss of hair, an overgrowth of hair, or a change in the color of the hair.

DISTURBANCES OF CONSCIOUSNESS

Coma.—Coma is a state of prolonged unconsciousness, somewhat resembling sleep, from which the patient cannot be aroused.

Temporary unconsciousness due to anemia of the brain is termed *syncope*. It may be recognized by the extreme pallor, weak pulse, and feeble heart-sounds. Coma may result from:

1. *Traumatism.*—This can be recognized only by the history or the local evidence of injury.

2. *Organic Disease of the Brain.*—The most common cause under this head is apoplexy, which may be recognized by the history, the age, the condition of the arteries, and by evidences of paralysis, such as stertorous breathing, unnatural relaxation or rigidity on one side of the body, conjugate deviation of the eyes, and a higher temperature in one axilla.

3. *Epilepsy.*—The coma of epilepsy is usually of short duration. It may be recognized by the history, by the bloody saliva, by the presence of scars on the tongue from previous attacks, and by the exclusion of other causes.

4. *Thermic Fever* (*Sunstroke*).—The temperature of the day or of the room in which the patient is found, the extremely high body-temperature, and the absence of other causes will usually prevent an error in diagnosis.

5. *Certain Drugs.*—Acute alcoholism and acute opium poisoning are familiar examples. In *alcoholism* the patient can usually be aroused by shouting in the ear, there is the odor on the breath, and other causes can usually be excluded.

In *opium-poisoning* the pupils are small, the respirations are slow, the temperature is normal or subnormal and there may be the odor of laudanum on the breath. The diagnosis will be aided by the exclusion of other causes.

6. *Uremia.*—In this condition there may be a urinous odor to the breath; the aortic second sound is often accentuated; the urine contains albumin and casts; the temperature may be above or below normal; the pupils are usually small and equal, and there is no evidence of other cause.

7. *The Infectious Fevers.*—The diagnosis is usually suggested by the clinical history. Pernicious malarial fever may produce sudden coma, and in this condition the examination of the blood affords conclusive evidence.

8. *Hysteria.*—The coma has the appearance of normal sleep; the patient can usually be aroused by painful stimuli, as pressure over the supraorbital nerve, or the suggestion of some painful procedure; the temperature, pulse and respiration are not usually altered, and there is often a history of other hysterical manifestations.

9. *Acetonemia.*—Diabetic coma may be recognized by the history, the fruity odor of the breath, the glycosuria, and the subnormal temperature.

Catalepsy.—This is a state of motor inertia, the limbs tending to remain for long periods in any position in which they are placed. During the attacks the patient is apparently insensible to external impressions. It occurs in hysteria, hypnosis, certain psychoses (melancholia attonita), and rarely in organic brain disease.

DISTURBANCES OF THE SPECIAL SENSES

The Eye.—*Myosis.*—Contraction of the pupil occurs in many conditions, notably in locomotor ataxia, paretic dementia, syringomyelia, early stages of increased intracranial pressure, paralysis of the cervical sympathetic, uremia, opium-poisoning, and old age.

Mydriasis.—Dilatation of the pupil is also observed in many conditions, notably in atrophy of the optic nerve, paralysis of the third nerve, severe pain, epileptic seizures, hysteric attacks, belladonna-poisoning, and in asphyxia from any cause.

Inequality of the Pupils.—Slight inequality may occur in health. Marked inequality (*anisocoria*) may be seen in ocular defects, in organic brain disease, in paretic dementia, in locomotor ataxia, in aneurysm pressing on the cervical sympathetic, and in unilateral paralysis of the oculomotor nerve.

Argyll-Robertson Pupil.—This is one that fails to respond to light, but still accommodates for distance. It is noted especially in locomotor ataxia and paretic dementia.

Conjugate Deviation of the Eyes.—This term is applied to the forcible deflection of the eyes to one side, the visual axes still remaining parallel. It is a common symptom in gross lesions, such as hemorrhage or tumor, of the motor centers or their tracts in the brain. When the lesion is cerebral and destructive the eyes are turned away from the palsied side (toward the lesion) and when the lesion is cerebral and irritative the eyes are turned toward the convulsed side (away from the lesion). In pontine lesions the deviations are exactly reversed.

Nystagmus (Tremor of the Eyeball).—It may be congenital, associated with defective vision, or due to a lesion in the posterior fossa. It is especially frequent in disseminated sclerosis and Friedreich's ataxia; it is not uncommon in miners.

Optic Neuritis or Papillitis.—An inflammatory affection of the intra-ocular end of the optic nerve. The term "choked disk" is used to designate the condition when it is accompanied with marked swelling. Its chief causes are: Tumor of the brain, cerebral meningitis, syphilis, toxic agents (lead and alcohol), infectious fevers, anemia, and Bright's disease.

Atrophy of the Optic Nerve.—As a primary affection it is most commonly observed in locomotor ataxia and paretic dementia. Secondary atrophy results from pressure of tumors, aneurysms, etc., on the optic chiasm. Consecutive atrophy is a sequel of optic neuritis.

The Ear.—*Tinnitus Aurium (Subjective noises in the Ear).*—This is observed in cerebral hyperemia and anemia, in diseases of the ear, in Ménière's disease, and after the use of certain drugs, like quinin and salicylic acid.

Hyperacusis of Hearing.—This is sometimes observed in hysteria, in facial paralysis, and in cerebral hyperemia.

Deafness usually depends upon disease of the ear itself.

PSYCHIC DISTURBANCES

Delusion.—A delusion is a faulty belief concerning a subject capable of physical demonstration, out of which the person cannot be reasoned by adequate methods for the time being (Wood).

A *systematized delusion* is one which the patient endeavors to defend by a process of reasoning more or less logical. Systematized delusions are especially observed in monomania.

An *unsystematized delusion* is one which the patient makes no attempt to justify; he asserts his belief without reason. The majority of delusions are unsystematized, and as such are observed in most forms of insanity.

A *fixed delusion* is one which the patient retains for a considerable length of time; it is frequently systematized. Fixed delusions are observed in monomania (paranoia), paretic dementia, hysteric insanity, and sometimes in melancholia.

An *expansive delusion* or a *delusion of grandeur* is one which exalts its possessor. The patient conceives that he is some noted personage, that he is worth millions of dollars, or that he is capable of performing certain marvelous feats. Expansive delusions are frequently observed in paretic dementia, mania, and hysteric insanity.

A *hypochondriacal delusion* is one which depresses its possessor. Thus, the patient may believe that he has committed the unpardonable sin, that he is being persecuted, or that he is the victim of some dread disease. Hypochondriacal delusions are frequently observed in melancholia, alcoholic insanity, and in some cases of monomania and paretic dementia.

Illusion.—An illusion is a perverted perception. Thus in delirium tremens the patient may transform every piece of furniture into a demon or reptile.

Hallucination.—An hallucination is a false perception, entirely subjective, and not based upon any knowledge derived from without. An individual who hears voices and sees objects when none exist is the subject of hallucinations.

Imperative Conception.—This is an idea which dominates the patient's actions to a greater or less extent, although its falsity is recognized.

A morbid impulse is an irresistible desire to commit an act which the patient knows to be wrong. It is usually the result of an imperative conception. *Kleptomania* is a morbid desire to steal. *Pyromania* is a morbid desire to set fire to buildings.

Delirium.—Delirium is a mental state characterized by a rapid flight of ideas that are incoherent and often unintelligible. It may result from:

Acute Delirium (*Bell's Mania*).—A disease arising without obvious cause, and characterized by an abrupt onset, active delirium, a constant repetition of certain phrases or acts, moderate fever, often a bullous eruption, and exhaustion. It generally ends fatally in the course of a few weeks.

Hysteria.—The history, the suggestibility and emotional instability of the individual, and the presence of other hysteric stigmata will aid in the diagnosis.

Infectious Fevers.—Careful physical examination and continued observation will almost usually disclose the true character of the case.

Uremia.—The urinous odor of the breath, the high arterial tension, the accentuation of the second aortic sound, and the presence of albumin and casts in the urine will suggest uremia.

Alcoholism.—The history, the appearance of the patient, the marked tremors, and frequently terrifying hallucinations will indicate alcoholism.

Inanition.—A form of delirium occasionally arises in the course of exhausting diseases. It is usually of short duration, and may be recognized by the circumstances under which it develops.

DISEASES OF THE BRAIN, SPINAL CORD AND NERVES

ACUTE CEREBRAL LEPTOMENINGITIS

Definition.—An acute inflammation of the pia mater and arachnoid.

Etiology.—(1) It may be a primary affection excited by the Diplococcus intracellularis (cerebrospinal fever) or by the pneumococcus. (2) It may be tuberculous, tubercle bacilli from a primary focus of disease elsewhere in the body reaching the meninges through the blood-vessels or lymph-channels. (3) It may follow injury, disease of the cranial bones, or otitis media (streptococcus, staphylococcus, pneumococcus). (4) It may be a complication of a specific fever—pneumonia, typhoid fever, erysipelas, influenza, etc. (the organisms exciting the primary infection or pyogenic bacteria).

Pathology.—The membranes are injected, cloudy, and more or less edematous. The subarachnoid space is distended with a seropurulent or purulent exudate. The substance of the brain may also be involved. The ventricles are often dilated and filled with turbid fluid. In some cases the process extends over the entire brain and even to the spinal cord; in others it is more or less localized to the convexity or base. The tuberculous form and that following middle-ear disease are usually basilar. In the tuberculous form, which is nearly always secondary, an infiltration of yellowish, gelatinous material is found at the base, especially about the optic chiasm. Small tubercles can usually be detected along the blood-vessels in the Sylvian fissures. The amount of fluid in the lateral ventricles is often considerably increased (acute hydrocephalus).

Symptoms.—The onset may be sudden or insidious. Headache, severe and persistent, is rarely absent. Vomiting is often a prominent symptom, especially in basilar meningitis. It frequently occurs independently of the presence of food in the stomach. The temperature is moderately high ([illegible]–104° F.) and very irregular. The pulse is sometimes infrequent (70 to 40 a minute). The bowels are constipated and the abdominal wall is retracted. Facial herpes is common in the meningococcic form. Symptoms of cerebral irritation soon develop. These comprise delirium, contraction of the pupils, photophobia, intolerance to sound, general hyperesthesia, Kernig's sign, muscular twitchings, and, perhaps, convulsions. When the exudate is sufficient in amount to exert marked pressure, paralytic phenomena develop. Palsies, gross or localized, take the place of convulsions; coma follows delirium; the pupils dilate and the eye-balls roll up; photophobia is replaced by blindness, and intolerance to sound by deafness. If the finger be drawn across the body, a bright-red line develops and lingers for some minutes; this is the *tâche cérébrale* of Trousseau. The pulse now becomes rapid, weak and irregular; the respiration often acquires the Cheyne-Stokes type, and the temperature falls, often becoming subnormal.

When the process involves the base, retraction of the head with rigidity of the back of the neck, optic neuritis, and paralysis of the cranial nerves are prominent symptoms. Fluid obtained by lumbar puncture is frequently turbid and may contain the organism provocative of the disease.

Diagnosis.—In *tuberculous meningitis* the onset is usually insidious, symptoms of ill health preceding the outbreak for days or weeks; the symptoms of a basilar involvement are marked; tuberculous lesions may be detected elsewhere in the body; tubercles are occasionally seen on the choroid, and finally fluid obtained by lumbar puncture may contain a large percentage of lymphocytes and tubercle bacilli.

Cerebrospinal Fever.—In this disease spinal symptoms—opisthotonos, pain in the back, and contractures—are usually

fever are usually due to tubercu
and only very rarely to menin

Prognosis.—The outlook is
purulent forms, and that follo
variably fatal. The duration is
or three weeks, but the menin
comes chronic and persists f
recovery occurs, blindness, pa
may remain as a permanent se

Treatment.—This should be
as that of cerebrospinal fever.
in a quiet, well-ventilated room.
to the head. In the robust, we
plied to the neck. Blisters are
be liquid. Constipation should
lessness, headache, and conv
chloral, phenacetin, or morphi
puncture are also of service. In
treatment should be instituted a
ear disease is the exciting fact
definitely localized, operation m

CHRONIC CEREB

mental deterioration, vertigo, muscular weakness, a low grade of optic neuritis, and occasionally nausea, vomiting, and tinnitus. Acute exacerbations are not infrequent, and are characterized by fever, severe headache, delirium, convulsions, and stupor.

Diagnosis.—*Cerebral Tumor.*—In tumor the symptoms are more severe and of a more focal character, and the optic neuritis is of a high grade.

Uremia may be recognized by the previous history, occurrence of chronic nephritis, albuminuric retinitis, etc.

Prognosis.—The outlook is very uncertain. A complete cure is sometimes obtained in syphilitic cases when specific treatment is instituted early.

Treatment.—In syphilitic meningitis mercury and potassium iodid should be used freely. In other instances courses of ergot and potassium bromid are occasionally useful. Applications of the thermocautery sometimes give relief. Tonics and somnifacients are frequently indicated.

CHRONIC CEREBRAL PACHYMENINGITIS

Definition.—Inflammation of the dura mater.

Etiology.—Inflammation of the external layer may result from injury, syphilis, or caries of the bone. Inflammation of the internal layer (hemorrhagic pachymeningitis) is most commonly met with in chronic insanities. Less frequently it follows trauma of the head or sunstroke, or occurs in chronic alcoholism, severe anemia, or chronic disease of the blood-vessels.

HEMORRHAGIC PACHYMENINGITIS

(Hematoma of the Dura Mater)

This condition is characterized by the formation of layers of delicate connective tissue extraordinarily rich in thin-walled blood-vessels from which the blood is prone to escape, producing hematomata of various sizes.

Symptoms.—In some cases there are no manifestations during life.

If the condition is marked, the following phenomena may be observed: Headache, failure of memory, impairment of intellect, stupor, contracted pupils, local convulsions, or palsies. The symptoms may alternately improve and grow worse over a long period. In grave cases, associated with extensive hemorrhagic effusion, the symptoms resemble those of cerebral apoplexy. The treatment is symptomatic.

CHRONIC HYDROCEPHALUS

(Congenital Internal Hydrocephalus; Water on the Brain)

Definition.—A condition in which there is an excessive accumulation of fluid in the ventricles of the brain.

Etiology.—The disease is either congenital or develops in the first few months of extra-uterine life. The etiology is obscure. In some cases the effusion appears to be the result of an inflammatory condition of the ventricular ependyma, while in others an occlusion of the communicating passages between the ventricles or between the ventricles and subarachnoid space is apparently the chief cause.

Pathology.—The head is large and round; the bones are thin and translucent; the sutures and fontanels are enlarged, and, if life has been prolonged, are filled with numerous Wormian bones. The convolutions of the brain are flattened and the sulci more or less obliterated. The ventricles are greatly distended with a watery fluid of low specific gravity, containing a trace of albumin. The ependyma is often thickened and roughened. Malformations are frequently observed, and probably result from the causes which induced the effusion.

Symptoms.—Sometimes the disease develops before birth, and the large head interferes with the delivery of the child. In other cases nothing peculiar is observed until the child is several months old, when the swelling of the head attracts the attention of the parents. The head assumes a globular shape; the fontanels and sutures remain open; the face becomes relatively small; the eyes protrude and are directed downward from the pressure of the fluid on the supra-orbital plates; the scalp is thin and stretched; the superficial veins are distended; and the hair is often scanty. In some cases the head is so heavy that the thin neck can no longer support it, and it falls forward on the breast.

As a rule, the intelligence is considerably impaired, but exceptional cases are marked by precociousness. Motor phenomena are frequently present; the reflexes are exaggerated; one or more of the members may be the seat of a spastic paralysis; and convulsions develop in many cases.

The duration varies in different cases. The large majority soon die of inanition, convulsions, or some intercurrent disease to which their reduced vitality makes them an easy prey; but in a few cases life is prolonged for many years.

Diagnosis.—Hydrocephalus must not be mistaken for *rachitic enlargement of the head.* In the latter the head is square instead of globular; the intelligence is good; there are no motor phenomena; and enlargements are usually detected at the ends of the long bones and at the junction of the cartilages with the ribs.

Prognosis.—The outlook is unfavorable. Occasionally arrest of the disease occurs spontaneously or results from aspiration of the fluid.

Treatment.—The treatment is unsatisfactory. In the majority of cases, beyond dietetic and hygienic measures and the occasional use of tonics, little can be recommended. Tapping of the ventricles or of the subarachnoid space in the lumbar region (Quincke's puncture), with gradual compression of the head by means of broad strips of adhesive plaster, sometimes affords temporary relief.

PARETIC DEMENTIA

(General Paralysis of the Insane; General Paresis; Chronic Meningo-encephalitis)

Definition.—A chronic inflammatory affection of the cerebral cortex, characterized by a change of disposition, failure of memory, mental exaltation, delusions of grandeur, tremors, epileptiform seizures, and paresis.

Etiology.—It occurs most frequently between the ages of thirty and fifty. It is much more common in men than in women. Syphilis is the chief, if not the only, etiologic factor. Mental strain, traumatism, alcoholism, and excesses of all kinds are auxiliary causes.

Pathology.—The membranes are opaque, thickened, and at places adherent to the brain substance. The cortex, especially of the frontal lobes, is more or less atrophied and increased in firmness. Microscopic examination reveals a marked increase in the neuroglia, with numerous spider cells, thickening of the vessel-walls, distention of the perivascular spaces with round cells, and degeneration of the ganglion-cells and atrophy of the nerve-fibers.

In some cases similar degenerative changes are observed in the spinal, especially in the dorsal columns (tabo-paralysis).

Symptoms.—The disease usually begins insidiously with a change in disposition: the industrious become slothful; the ambitious, apathetic; the chaste, dissolute; the liberal, parsimonious; the complaisant, churlish; and the truthful, false. The energy relaxes, the judgment weakens, and the memory fails. As the faculties become impaired, a peculiar egotism and mental exaltation develop; the patient becomes boastful, loquacious, and easily provoked to furious outbreaks. The failure of memory is early noted in writing, by the use of wrong letters and the suppression of syllables. At this time motor phenomena may be observed; the tongue trembles when it is protruded; the speech is slow, hesitating, and indistinct; the pupils are often unequal; and the gait is somewhat shuffling.

The most characteristic psychic symptom of fully developed paretic dementia is the delusion of grandeur, which is shown in the patient's exaggerated estimate of his social or political status, wealth, strength, or intellectual power. The mind is usually serene and cheerful, but periods of profound depression are not infrequent. The sensibilities are blunted, and the animal nature is emphasized. The mind becomes more and more involved; there is extreme indifference to all that transpires; the appetite is voracious, and in eating the patient bolts his food and soils his clothes. The tremor of the tongue increases, and spreads to the lips and other parts of the face; the speech is indistinct and "scanning;" the pupils fail to respond to light, but still accommodate for distance (Argyll-Robertson pupil); and the reflexes are usually increased, though they may be lost. Seizures of an epileptiform or apoplectiform character are not uncommon. The cerebrospinal fluid contains a considerable amount of albumin (globulin) and an excess of lymphocytes and yields a positive Wassermann reaction.

In the final stage mental power is almost obliterated; the health fails; the bladder and rectum become unretentive; the gait is more unsteady; and at last the patient is unable to leave his bed. Death usually results from exhaustion or intercurrent disease.

Diagnosis.—The insidious change in disposition, failure of memory, indistinct speech, tremors, Argyll-Robertson pupil, delusions of grandeur, positive Wassermann reaction, and spinal lymphocytosis are the diagnostic features.

Cerebral Syphilis.—Severe headache, worse at night, somnolence, localized paralyses, especially of the cranial nerves below the third, early emaciation, and absence of the Argyll-Robertson pupil, of generalized tremors, and of delusions of grandeur are in favor of cerebral syphilis.

Prognosis.—The outlook is bad. The course is not uniform: occasionally there are remissions, or lucid intervals, which last for several months or even years. The average duration is from two to three years.

Treatment.—This is chiefly hygienic and dietetic. The avoidance of mental and physical excitement is imperative. Antisyphilitic treatment should be given a thorough trial. Some clinical improvement has been obtained from intraspinal injections of serum separated from the patient's blood an hour after the *intravenous* administration of salvarsan. Swift-

at birth or in the first few year
with atrophy and sclerosis of
cephalus.

Pathology.—After death on
found: Porencephalus (cystic c
and sclerosis of the convolut
meningeal hemorrhage. The p
is not definitely known, but it
in *congenital* cases it is usually
by difficult labor; and in *infan*
cephalitis, hemorrhage, thromb
an antecedent infection.

Symptoms.—*In the hemipleg*
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After a few hours or days these
the child is left paralyzed on o
instances the paralysis ultimat
is restored to health, but in t
persists and is followed by se
epilepsy, and choreiform or athe
members are very common sequ

The *diplegic* or *paraplegic* f

by the mouth or rectum. In congenital cases, when there are bulging of the fontanels, asphyxia, and convulsions, operation should be considered. The paralysis usually resists treatment; but subsequent rigidity may be lessened by massage and passive movements, and the deformity by mechanical appliances. Division of the posterior nerve-roots has been practised with some success in overcoming spasticity in the legs.

CEREBRAL HYPEREMIA

(Congestion of the Brain)

Etiology.—*Acute congestion* results from exposure to the sun; from the use of certain drugs, like alcohol and nitroglycerin; from excessive brain-work; or from some reflex disturbance, as gastric irritation.

Chronic congestion results from some local obstruction to the return of blood from the brain, as by a tumor in the neck; from obstruction to the general circulation, as in chronic heart and lung disease; from the suppression of some habitual discharge, as the menstrual flow at the menopause; or from some general cause, such as prolonged anxiety, overwork, excesses, irregular living, etc.

Symptoms.—*Acute Form.*—The chief symptoms are: Intense headache; vertigo; intolerance to light and sound; restlessness; tinnitus aurium; and sleeplessness, or sleep disturbed by unpleasant dreams.

Chronic Form.—This is characterized by vertigo; dull headache; failure of memory; irritability; inability to concentrate the thoughts; and disturbed sleep. The symptoms grow worse when the recumbent posture is assumed. Ophthalmoscopic examination reveals retinal hyperemia. In marked cases there may be exacerbations closely resembling apoplexy, in which there is unconsciousness, followed by transitory paresis.

Prognosis.—The prognosis depends upon the cause; if this can be removed, the prognosis is favorable.

Treatment.—*Acute Congestion.*—The patient should be placed in a darkened, well-ventilated room. The head and shoulders should be slightly elevated. An ice-bag should be applied to the head. In some cases leeches or wet-cups may be applied to the neck. Sedatives like bromid of potassium and aconite sometimes do much good. Hot foot-baths are often serviceable. If there is constipation, it should be relieved by a brisk saline purge.

In *chronic cases* the cause should be ascertained, and, if possible, removed. The habits of the patient must be regulated. The diet must be light and nutritious. Constipation must be relieved by diet or by the occasional use of a saline laxative. Sedatives like bromid of potassium and aconite are useful. In the apoplectiform attacks venesection is indicated.

CEREBRAL ANEMIA

Etiology.—General cerebral anemia as a *chronic affection* may result from cardiac disease, especially aortic stenosis. It may be associated with general anemia. It may be due to atheromatous obstruction of the arteries.

Overwork, prolonged emotional excitement, irregular living, and excesses are also said to predispose.

As an *acute condition* it exists in syncope and shock; after hemorrhage; after the sudden withdrawal of fluid from the abdominal cavity; and after ligation of the carotid artery.

Symptoms.—*Acute Form.*—Pallor of the face, vertigo, confusion of ideas, ringing in the ears, dimness of vision, dilatation of the pupil, nausea, and a tendency to yawn. In extreme anemia there may be convulsions and coma.

The *chronic form* is characterized by vertigo, headache, disturbed sleep, intolerance to light and sound, irritability of temper, failure of memory, inability to concentrate the attention on one subject, a tendency to syncope, and extreme lassitude. The symptoms improve when the patient lies down.

Diagnosis.—Cerebral anemia closely simulates *cerebral congestion*, but in the latter there is no tendency to syncope; the symptoms grow worse when the patient lies down, and the pupils are usually contracted instead of dilated.

Prognosis.—The prognosis depends on the cause; if this can be removed, the prognosis is favorable.

Treatment.—In acute cases diffusible stimulants like nitroglycerin, ammonia, and alcohol are indicated. In chronic cases the cause should be ascertained, and, if possible, removed. When it is due to general anemia, iron, arsenic, and quinin are useful remedies. When dependent on valvular disease, rest and the use of digitalis, strophanthus, or strychnin are the chief remedial measures.

CEREBRAL HEMORRHAGE

(Cerebral Apoplexy)

Etiology.—The affection is most commonly met with in persons over forty, in whom the blood-vessels are atheromatous, but it may occur in childhood or infancy. All causes that lead to degeneration of the arteries, such as rheumatism, gout, syphilis, alcoholism, and Bright's disease, predispose to it. Sufferers from chronic Bright's disease are very liable to apoplexy on account of the association of cardiac hypertrophy with arterial degeneration. Heredity predisposes, inasmuch as members of certain families are particularly prone to sclerosis of the vessels.

The attack may be precipitated by mental or physical excitement, alcoholic excess, or some reflex disturbance, as gastric irritation. In children it may be excited by a paroxysm of whooping-cough or by a convulsion. Occasionally, it occurs after diphtheria or scarlet fever, the toxins of these diseases producing degenerative changes in the arterial walls.

Pathology.—In children the hemorrhage is most commonly cortical; in adults it is usually within the brain-mass.

The blood-vessels are usually atheromatous, and are sometimes the seat of miliary aneurysms. The hemorrhage varies greatly in quantity; sometimes it is small—merely a capillary oozing; at other times it may flood an entire hemisphere. Its most common seat is the internal capsule—the motor highway between the optic thalamus and the corpus striatum. In recent hemorrhages the clot is dark and soft, and the surrounding tissue stained and more or less lacerated. If the hemorrhage has not been very copious, the clot loses its color, shrinks, and is finally absorbed, and the damaged cerebral fibers are replaced by proliferated connective tissue, which contracts and forms a scar more or less pigmented with hematoidin. In other cases, instead of a scar, a cyst is formed enclosing a clear, straw-colored fluid.

Large effusions are followed by secondary changes, which are systemic and extend in the direction in which the affected nerve-fibers transmit impulses; that is, toward the periphery, if the fibers are motor, and toward the nerve-centers if they are sensory. After an extensive lesion of the internal capsule secondary degeneration of the motor tracts soon begins and may be traced downward into the spinal cord.

Symptoms.—*Prodromal symptoms* indicating cerebral congestion frequently precede the attack; these are headache, vertigo, disturbed sleep, tinnitus aurium, and, perhaps, a sense of numbness or weakness on the side that is to be affected. Persistent vomiting sometimes precedes the hemorrhage.

The Attack.—In many cases the patient falls suddenly unconscious without previous warning. The face is flushed; the eyes are injected; the lips are blue; the breathing is stertorous; the pulse is full and slow; the temperature is at first subnormal from shock, but later it is elevated from irritation; and the urine and feces may be passed involunt
Convulsive seizures are not infreque
irritation transmitted to the und
ven while the patient is comat

detected. The head and eyes may be strongly rotated toward the side of the hemorrhage (conjugate deviation); one cheek often flaps more than the other; the pupils may be unequal; any movements that the patient may make are restricted to the sound side; when the affected arm is raised and let fall, it drops lifeless; and occasionally the temperature is higher in the axilla of the paralyzed side. In grave cases the patient does not awake from the coma; the pulse grows feeble; the respirations acquire the Cheyne-Stokes type; the reflexes are abolished; mucus collects in the throat and produces a rattling sound; the temperature rises to 103° to 104°; and death results after the lapse of a few hours or one or two days.

In certain cases the paralysis rapidly sets in, but unconsciousness develops gradually and does not become complete for twenty-four hours (ingravescent apoplexy). In other cases of cerebral hemorrhage loss of consciousness is very transient or wholly wanting.

Subsequent Symptoms.—If the attack does not prove fatal, consciousness is usually restored in from twelve to forty-eight hours, and if the hemorrhage is in its usual location, there remains a hemiplegia on the opposite side. The muscles of the upper part of the face and thorax, however, usually escape, because they are accustomed to act in unison with their fellows on the opposite side, and such muscles appear to be innervated from both sides of the brain. When the tongue is protruded, it deviates toward the paralyzed side. The deep reflexes are exaggerated on the affected side and irritating the sole of the foot causes extension of the great toe (Babinski's sign). Aphasia is common with right hemiplegia. There is no tendency to rapid wasting of the affected muscles. Sensation is unimpaired unless the entire posterior limb of the internal capsule is involved, when there is hemianesthesia with the hemiplegia. The gait is peculiar; in walking the patient supports the paralyzed arm and swings the leg forward by a rotary movement imparted to it by the tive of permanent disability.

Mental symptoms, especia
loss of emotional control, fre

Diagnosis.—The coma of
from that of *uremia*, *opium*
stroke. The age of the patie
signs of paralysis; the differe
axillæ; and the absence sympt
will usually prevent an error

Embolism.—This usually o
monly associated with valvul
toms are rarely present; the
strong; disturbances of temp
marked.

Thrombosis.—This also pro
velopment is usually gradual;
and the temperature and breat

Hemiplegia from Other Caus
brain may produce hemiplegia
ally and is usually associated
such as persistent headache,
disc, etc.

Hysteric

respiration are unfavorable phenomena. If the attack does not prove fatal, there is always danger of recurrence, since the etiologic conditions still remain.

Treatment.—*Prophylaxis.*—Patients predisposed to apoplexy should lead a quiet life, free from mental and physical excitement. The diet should be nutritious, but easily digestible. Constipation should be relieved by the occasional use of a saline laxative. To secure a free return of the blood from the brain the clothes at the neck should be loose.

The Attack.—The head and shoulders should be slightly elevated, and an ice-bag applied to the head. Croton oil (1 to 3 drops) in a little glycerin or olive oil may be placed on the back of the tongue to secure prompt catharsis. If the pulse is strong, venesection is indicated and should be continued until the pulse softens. Bleeding cannot undo the damage already done, but by relieving cerebral congestion, it may serve to arrest bleeding that is still in progress or to prevent an early recurrence. On the other hand, when the face is pale and the pulse feeble, stimulants, like ammonia, ether, and camphor, should be given very cautiously. If collections of mucus interfere with breathing, the patient should be gently turned on his side and the mucus removed.

To prevent the formation of bed-sores the position should be frequently changed and the parts subjected to pressure thoroughly cleansed.

Subsequent Treatment.—Even in the mildest cases the patient should not be allowed to leave his bed for two or three weeks, and during this time the diet should be light and unstimulating. After the acute symptoms have entirely disappeared, which will rarely be earlier than ten days or two weeks after the attack, massage should be systematically practised. It aids in the restoration of power and in the prevention of contractures. After the lapse of three or four weeks triweekly applications of the faradic current may be of service. Strychnin is often given at this time, but it probably exerts no other influence than that of a general tonic. In some cases warm saline baths (90°–95° F.) combined with passive movements prove useful adjuvants.

OBSTRUCTION OF THE CEREBRAL ARTERIES

(Embolism; Thrombosis)

Etiology.—*Cerebral emboli* may be derived from the valves of the heart in endocarditis; from an atheromatous plate in the aorta; or from a thrombus in the heart or in the sac of an aneurysm. Obstruction from embolism may occur at any age, but it is far more commonly observed in young adults than at the extremes of life.

Cerebral thrombi are usually caused by atheroma or syphilitic endarteritis. They are usually observed in advanced years, but those dependent on syphilis frequently occur in early adult or middle life.

Pathology.—*Emboli* are most frequently found in a branch of the left middle cerebral artery. If the artery obstructed is a large one, infarction of the brain with softening ensues. If the area affected is small, absorption of the dead tissue usually follows, with the formation of a cicatrix. Infective emboli give rise to abscesses.

Thrombi are usually found in the basilar, middle cerebral, or vertebral arteries, and produce the same lesions as emboli.

Symptoms.—*An embolus* lodging in the middle cerebral artery usually causes abrupt hemiplegia and frequently aphasia. There may be no prodromes, and consciousness may be preserved during the seizure.

If the basilar artery is obstructed, there may be extensive paralysis on both sides of the body, and later symptoms of bulbar disease—namely, paralysis of the lips, pharynx, and esophagus, disturbance of the heart, and Cheyne-Stokes breathing.

In *thrombosis* the symptoms are similar to embolism, but they develop more slowly, and are frequently preceded by prodromes indicating disturbed cerebral circulation, such as headache, vertigo, disturbed sleep, failure of memory, numbness and tingling in the limbs to be affected. There is a marked tendency to recurrence of attacks.

Subsequent Symptoms.—In both embolism and thrombosis, if the artery obstructed has been large, the paralysis is likely to persist and to be followed by symptoms of cerebral softening—namely, failure of memory, vertigo, headache, disturbed sleep, irritability, and finally dementia.

Diagnosis.—The differential diagnosis between cerebral embolism or thrombosis and *hemorrhage* has already been considered (see p. 464).

Prognosis.—The prognosis is always grave; unless the symptoms are slight, complete recovery is rare.

Treatment.—After obstruction from embolism the patient should be kept at absolute rest for a week or two, and subsequently the paralysis treated as after apoplexy. In thrombosis treatment is rarely of avail; in syphilitic cases, however, active antiluetic treatment should be instituted.

MORBID GROWTHS IN THE BRAIN

(Tumors of the Brain)

Etiology.—The etiology of brain tumors is obscure. Males are more frequently affected than females. No age is exempt, but the majority of brain tumors occur between the ages of thirty and fifty. Occasionally the history of some remote injury is obtainable. Heredity predisposes to the extent that it favors the development of tubercle and cancer.

Varieties.—Tubercle, gumma, glioma, aneurysm, cysts, sarcoma, and carcinoma are the most common varieties. Less frequently fibroma, psammoma, and lipoma are observed.

Pathology.—*Tuberculous tumors* or *tyromata* vary in size from that of a pea to that of an egg; they may be single or multiple, and are usually observed in the young.

Gumma.—This appears as a round, yellow, caseous mass, and is nearly always on the surface of the brain, into which it grows from the overlying membranes. It is usually met with between the ages of thirty and forty.

Glioma.—This tumor is found almost exclusively in the brain. It arises from the neuroglia, and may be soft, like brain-substance, or firm, like fibrous tissue. It is chiefly met with in the young.

Aneurysm.—Encephalic aneurysm may be single or multiple. Miliary aneurysms of small vessels frequently excite apoplexy. The most common seats of large aneurysms are the middle cerebral, basilar, and internal carotid arteries.

Cysts.—These are usually congenital (porencephalus) or result from hemorrhage, but sometimes they result from the Tænia echinococcus (hydatid cyst) or Tænia solium (Cysticercus cellulosæ).

Fibrosarcoma (Endothelioma).—This is usually a circumscribed tumor, and commonly grows from the membranes. It is generally primary.

Carcinoma.—This is nearly always secondary and multiple.

Symptoms.—*General Symptoms.*—(1) Headache is rarely absent; it is sometimes localized and associated with tenderness on pressure. (2) Vomiting is a common symptom, especially in tumors at the base of the brain; it is often unassociated with nausea, and does not relieve the attending headache. (3) Choked disc is present in about 80 per cent. of the cases. (4) Vertigo is often marked, especially in tumors of the basal ganglia and cerebellum. (5) Convulsions, local (Jacksonian epilepsy) or general, occur in about 50 per cent. of all cases. (6) Psychic phenomena—failure of memory, depression of spirits, irritability of temper, and emotional states—are not infrequently present. Insomnia, changes in the rate and rhythm of the pulse, polyuria, and glycosuria are occasional symptoms.

Focal Symptoms.—These depend entirely upon the location of the tumor. The following are the chief localizing symptoms:

Prefrontal Region.—Mental torpor, irritability, loss of memory, and change in disposition frequently appear. Motor agraphia and aphasia may result from compression of the second and third frontal convolutions.

Motor Region (*the Ascending Frontal Convolution*).—When the tumor irritates the centers, local convulsions develop; when it exerts enough pressure to destroy function, paralysis results.

Posterior Portion of the Third Frontal Convolution (*Left Side*).—Motor or ataxic aphasia is a characteristic symptom.

Temporal Lobe.—Growths involving the first and second convolutions of left side may produce word-deafness. Lesions of the uncinate gyrus cause so-called uncinate seizures, which are characterized by disagreeable subjective sensations of smell or taste, chewing or swallowing movements, and a dreamy state of consciousness.

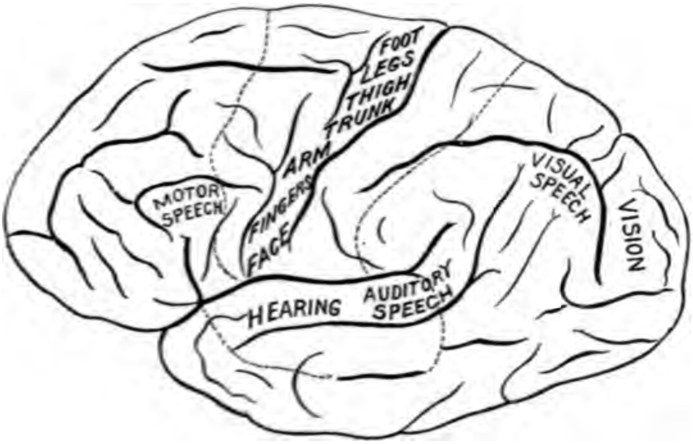

FIG. 17.—Functional area of the cerebral cortex, left hemisphere.

Angular Gyrus.—A tumor of the angular gyrus, if deep-seated, may involve the optic radiation and cause hemianopsia, and, also, if on the left side, word-blindness.

result

Internal Capsule.—Les
the posterior limb cause
and lesions of the poste
opposite side.

Corpus Striatum.—Larg
pressure on the internal ca

Optic Thalamus.—Tumo
anesthesia, often associated
and astereognosis, slight he
movements—all on the opp
smile and weep voluntarily,

Corpora Quadrigemina.—
cles (ophthalmoplegia) and
symptoms.

Crus Cerebri.—Tumors in
the third nerve on the side
the other side.

Pons.—Pontile growths m
hemianesthesia on the side op
sis of the cranial nerves, espec
of the lesion.

Corpus Callosum.—Tumors i

(adiadococinesis), and disordered phonation. Nystagmus is also a frequent symptom.

Diagnosis.—*The Character of the Growth.*—This cannot always be determined. The early age, the rapid progress, and the family history may suggest *tubercle*. The early age, slow progress, and mild pressure-symptoms may suggest *glioma*. The history, age, and concomitant symptoms will indicate *syphilis*. The presence of a primary growth will lead to the diagnosis of *cancer*. The presence of a thrill, bruit, and marked tinnitus and the absence of optic neuritis may suggest aneurysm.

Abscess.—Cerebral tumor must be distinguished from abscess. The latter usually results from traumatism or is secondary to a focus of suppuration in some other part of the body; its progress is usually more rapid; optic neuritis is less common; and there is often febrile disturbance, with leukocytosis.

Chronic Meningitis.—In this affection the symptoms indicate a diffuse lesion; disturbances of temper, memory, and sleep are more marked; optic neuritis is not frequent.

Prognosis.—The outlook is always grave. If the tumor is not gummatous and is not suitable for operative interference, the prognosis is absolutely unfavorable. The duration is from a few months to several years.

Treatment.—As there is always a possibility that the tumor is syphilitic, mercury and iodids should be tried in every case the nature of which is in the least uncertain. Operative interference must be considered when the tumor is localized and situated in the cerebral cortex or cerebellum. Less than 10 per cent. of brain tumors are favorable for removal. Growths in the motor region offer the best chances of success. Even if removal is out of the question "decompression" (removal of a piece of bone with opening of the dura) may relieve the pressure symptoms, especially severe headache and choked disc.

Except in *syphilitic* growths non-operative treatment is

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in pulmonary abscess, hep
ditis. (4) It may follow on

Pathology.—The abscess
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filtrated. In the acute form
long-standing cases the pus is
sac. The temporosphenoidal
the most frequent seats. A
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Symptoms.—Abscesses fol
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headache, delirium, convulsio

In chronic cases the *gener*
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ment, vertigo, vomiting, stup
strength. The temperature i
but not rarely it is normal
nomena vary with the

symptoms indicate involvement of an accessible region like the motor area, temporosphenoid lobe, or cerebellum, operative interference offers considerable hope of success.

Treatment.—If the abscess is located in one of the regions specified, the skull should be trephined and the pus evacuated. In other cases the application of wet cups to the neck, of ice-bags to the head, and the internal use of opium, bromid of potassium, or of chloral may temporarily relieve the distress.

APHASIA

Definition.—An inability to express ideas in speech or equivalents of speech or to comprehend speech or equivalents of speech.

Varieties.—Aphasia is divided into *motor* and *sensory*. Each of these is subdivided into *cortical* and *subcortical*, according as the lesion is the center itself or in the tracts communicating with the center. Sensory aphasia is further classified as *visual* and *auditory* aphasia.

Motor Aphasia.—This is an inability to express thought in words. When the lesion is in the third frontal convolution (*cortical motor aphasia*) the power of silent talking and reading are lost as well as that of articulate speech. When the lesion is in the adjacent tracts which transmit speech impulses to the articulatory muscles (*subcortical motor aphasia*), the power of articulation alone is lost. This is the most common form of aphasia.

Sensory Aphasia.—This is an inability to understand printed or written words (*visual aphasia* or *word-blindness*), or to understand spoken words (*auditory aphasia* or *word-deafness*). The lesion is in the angular gyrus, where visual word memories are stored, or in the first temporal convolution, where auditory word memories are stored, or in one of the incoming (*subcortical*) tracts of special sense.

In *cortical visual aphasia* the patient cannot read aloud or to himself, nor can he write (*agraphia*) spontaneously or

though word-deaf, can speak
write.

Pathology.—The lesions t
fold; the most important ar
pressed fracture, embolism,
localities that correspond to
In right-handed subjects the
brain; in the left-handed it
side. Aphasia is not always
occur as a transient conditio
sudden fright, in the conval
after epileptic seizures, and in

Diagnosis.—Aphasia must
The latter condition is an ina
not lost in aphasia; moreover
ent upon some abnormality
leading thereto.

Prognosis.—This depends
apoplexy the prognosis sho
softening it is absolutely un
velops in the young, the outlo

Treatment.

as a part of cerebrospinal meningitis. As a primary disease, without involvement of the cranial meninges, it is rare. It occasionally follows one of the infectious fevers, traumatism, or exposure. In some instances it is tuberculous.

Pathology.—*Acute Form.*—The membranes are opaque, thickened, congested, and adherent. The fluid in the arachnoid space is increased. In very acute cases there is more or less purulent infiltration. The periphery of the cord is always involved.

Chronic Form.—The membranes are thick and fused into one homogeneous fibrous mass.

Symptoms.—*Acute Form.*—The disease may begin with a chill, followed by a moderate fever. There is intense pain in the back, radiating along the course of the nerves. The back is tender. The spinal muscles are rigid and contracted—sometimes so much so as to induce opisthotonos. The reflexes are increased. When the exudate is sufficient to make considerable pressure on the cord, paralytic phenomena develop, such as slight anesthesia and paresis of the limbs.

There are no cerebral symptoms unless the meninges of the brain are involved.

Diagnosis.—*Myelitis.*—In this affection the pain is less severe; there is less tendency to spasm; paralysis and anesthesia are more marked; the bladder and rectum are early involved; and the formation of bed-sores is common.

Tetanus.—The presence of a wound; the absence of fever; the early involvement of the jaw; and the absence of marked tenderness in the back will suggest tetanus.

Prognosis.—The outlook is grave. Recovery sometimes follows, but rarely without partial paralysis.

Chronic Leptomeningitis.—This is characterized by pain in the back; stiffness of the muscles; hyperesthesia and paresthesia of the limbs, but rarely anesthesia; some loss of power; and exaggerated reflexes.

Treatment.—The treatment is that of cerebrospinal meningitis.

Pathology.—The membranes ar
vessels are dilated; and the spinal fl
membranes are glued together an
mass. The cervical region is most c
may extend to the cord and periphe

Symptoms.—These include shar
shoulders and arms, muscular weakn
and the peculiar deformity known
plegia, with exaggerated reflexes and
ensue from pressure on the spinal
several years.

Diagnosis.—*Chronic Poliomyelitis.*
thesia will separate poliomyelitis fro

Multiple Neuritis.—In this affectio
and more marked in the extremities,
pressure.

Syringomyelia.—In this affection t
neck and arms.

Prognosis.—This depends on th
volvement is slight or is due to
guardedly favorable.

Treatment.—Counterirritation sh
quent blisters or the actual cautery.
tered for its absorbent effect, and in s
in conjunction with some mercurial.

as *disseminated myelitis* if there are a number of discrete foci of disease, and as *poliomyelitis* (see p. 397) if the lesions are confined to the gray matter.

Etiology.—The disease may be a sequel if one of the acute infections; it may occur after trauma; it occasionally develops during the puerperium; it is sometimes secondary to disease or injury of the spine (*compression myelitis*); very frequently, however, it is the result of syphilis.

Pathology.—In early cases the affected area is soft and hyperemic; in late cases it is firm and pale (sclerotic). In the inflammatory form microscopic examination reveals degeneration or actual disintegration of the nervous elements, dilatation of the blood-vessels, and perivascular round-cell infiltration; or if the disease has been of long duration, an overgrowth of neuroglia tissue in place of the nervous elements which have been destroyed (sclerosis). In the syphilitic form the first change is a gummatous infiltration of the cord; or, more commonly, an area of ischemic necrosis (softening), the result of chronic endarteritis, and, perhaps, obstructive thrombosis. Ultimately, as in other forms of myelitis, proliferated neuroglia tissue is seen in place of cells and fibers that have undergone liquefaction.

Symptoms.—*Transverse Myelitis.*—The onset is acute and marked by numbness in the limbs, and sometimes by pain in the back and a girdle sensation. Constitutional symptoms: Chilliness, malaise, fever, and vomiting are also present in the inflammatory form. Paralysis and anesthesia for all forms of sensation quickly develop in the parts below the level of the lesion. The paralysis is flaccid and atrophic in the muscles supplied by the diseased segments, and flaccid or spastic in the muscles innervated by segments below the level of the disease, according as the latter completely or partially interrupts the conductivity of the cord. In the lower limbs, even if the paralysis is flaccid at first, it usually becomes spastic in the course of a week or two if the patient survives. The sphincters of the bladder and rectum are nearly always affected, sexual power is abolished, and bed-sores often develop with great rapidity over the buttocks and heels. Atrophy and reactions of degeneration occur in the muscles supplied by nerves arising from the diseased segment.

The outlook is always serious. More or less disability usually remains after the subsidence of the acute symptoms. Complete recovery may occur, however, in mild cases. Not rarely death ensues from cystitis and pyelonephritis, bed-sores, or paralysis of the respiratory musculature.

Acute ascending myelitis is characterized by progressive loss of motion and sensation beginning in the legs and rapidly ascending until the respiratory musculature is affected. Death frequently occurs at the end of a few days from asphyxia, or at a later period from cystitis, bed-sores, or hypostatic pneumonia.

Disseminated myelitis is rare. It produces palsy of isolated groups of muscles and irregularly distributed areas of anesthesia. In the later stages of the disease the clinical picture may be that of multiple sclerosis.

Compression myelitis is characterized by localized and deep-seated pain in the back, rigidity of the spinal column, angular deformity, stabbing pains radiating to the limbs or around the trunk (girdle pains), spastic paralysis below the level of the lesion, impairment of sensation, and disturbance of the sphincters.

Chronic Myelitis.—This term is often applied to the chronic stage of acute myelitis and to compression paraplegia, the result of vertebral disease or spinal tumor. As a slowly progressive condition it is occasionally met with in syphilis. The chief feature is slowly developing spastic paraplegia with a certain amount of sensory disturbance, usually paresthesia, and of sphincter trouble. The last not rarely precedes the other symptoms by several months.

Diagnosis.—*Acute Poliomyelitis.*—In this disease the bladder and rectum are not affected and there is no anesthesia.

Landry's Disease or Acute Ascending Paralysis.—In this affection there is an absence of sphincteric disturbance and of muscular wasting, and if sensory disorders are present, they are slight.

Multiple Neuritis.—The sphincters are not affected; bed-sores are rare; the pains are referred to peripheral parts of the limbs, and the nerve-trunks are tender on pressure.

Meningitis.—In this disease hyperesthesia, pain, rigidity of the back, tonic muscular spasms, and Kernig's sign are prominent in the clinical picture, while paralysis is less pronounced, the sphincters escape, and bed-sores are usually absent.

Hemorrhage into the Cord.—In this condition the paralysis develops abruptly.

Treatment.—If possible, the patient should be placed on a water-bed or air-bed. Counterirritation should be avoided on account of the danger of bed-sores. Cold, however, in the form of Chapman's ice-bags may be applied to the spine. Daily warm baths (90° F.) lasting about ten minutes are useful. When there is suspicion of syphilis, mercury or iodids should be given a thorough trial.

Every precaution should be taken against the development of bed-sores. Frequent change of the patient's position, absolute cleanliness of the parts subjected to pressure, and bathing with alcohol and water will do much toward obviating this complication. Retention of urine must be met by systematic catheterization under the most strict aseptic precautions. When there is constant incontinence, a carefully adjusted urinal should be employed.

Any tendency to cystitis will call for daily irrigation of the bladder with a solution of boric acid or other mild antiseptic solution. After the acute symptoms have entirely subsided, passive movements, massage, and electricity should be employed to maintain the nutrition of the affected muscles and to prevent fixation of the joints.

CHRONIC ANTERIOR POLIOMYELITIS—PROGRESSIVE SPINAL MUSCULAR ATROPHY

Definition.—A chronic disease characterized anatomically by atrophy of the nerve cells in the anterior gray horns of the spinal cord, and manifested clinically by a progressive wasting of the muscles and a corresponding loss of power.

Etiology.—The disease is more common in males than in females. It occurs most frequently in adults between the ages of twenty and fifty. The exact cause of the affection is unknown, but it is probable that the cells degenerate prematurely owing to inherent weakness (abiotrophy).

Pathology.—Microscopic examination of the cord reveals atrophy or complete disintegration of the ganglion cells in the anterior cornua and an overgrowth of the neuroglia. The anterior nerve-roots, peripheral motor nerve-fibers, and affected muscles also show degenerative atrophy. In addition to these lesions, there is often sclerosis of the anterolateral white tracts (amyotrophic lateral sclerosis).

Symptoms.—The onset is insidious. The muscles of the hand usually suffer first. The thenar and the hypothenar eminences and the interosseous muscles become more flaccid than normal and gradually waste. Accompanying the atrophy there is a corresponding loss of muscular power. When the interossei no longer afford opposition to the long flexor and extensor muscles, the hand assumes a claw-like position (*main en griffe*), which is quite characteristic. Fine fibrillary tremors or twitchings are almost invariably present in the affected muscles. After the lapse of months, perhaps years, the wasting and paresis spread to the muscles of the shoulder and arm, and then to the neck and trunk. The legs are usually not involved until late, and often escape entirely. Occasionally, however, the disease begins in the lower extremities or back, but this is rare. In the late stages the patient may be reduced to a mere skeleton. In many cases the process invades the medulla, in which case the symptoms of *bulbar palsy* are superadded.

There may be some complaint of coldness or of dull pain, but sensation is not impaired. The deep reflexes are lost in the affected limbs and the paralyzed muscles remain flaccid. The reactions of degeneration are sometimes present, but more often there is simply diminished response first to the faradic and then to the galvanic current. The sphincters are not involved.

Diagnosis.—Chronic poliomyelitis must be distinguished from other conditions causing slowly progressing atrophy and weakness, such as amyotrophic lateral sclerosis, muscular dystrophy, multiple neuritis, and syringomyelia.

Amyotrophic Lateral Sclerosis.—In this disease the atrophy is associated with spastic symptoms and the tendon-reflexes are exaggerated.

Muscular Dystrophy.—This is commonly an hereditary or a family affection. It occurs in childhood and attacks primarily large muscles (calf, shoulder girth, or face). There is no fibrillary twitching.

Multiple Neuritis.—In this disease the paralysis precedes the atrophy. Sensory symptoms are usually prominent, and there is often tenderness along the nerve-trunks.

Syringomyelia.—In this affection the atrophy is accompanied by exaggerated reflexes, peculiar sensory disturbances, and trophic changes in the skin and joints.

Prognosis and Treatment.—The course of the disease is very slow and occasionally marked by remissions. Death may result from bulbar paralysis or intercurrent disease. Treatment is of no avail.

PRIMARY SPASTIC PARAPLEGIA

(Lateral Sclerosis; Anterolateral Sclerosis)

Definition.—A very rare disease, characterized by gradual loss of power, marked exaggeration of the reflexes, and a spastic condition of the muscles, without atrophy or sensory disturbances.

Etiology.—The etiology is obscure. The disease usually develops between the ages of twenty and forty. Both sexes are equally affected.

Pathology.—A primary degeneration of the lateral pyramidal tracts (terminations of the upper motor neurons) is assumed to be the anatomic cause of the disease.

Symptoms.—Loss of power is usually the first symptom. This begins in the lower extremities and increases very slowly. The knee-jerk is exaggerated, and in most cases ankle-clonus can be elicited. When put in use, the muscles become stiff or spastic, and when the disease is fully developed, the gait is peculiar. In walking the knees are drawn together, the legs drag behind, and the toes catch the ground. The muscles do not waste, sensory and trophic disturbances are absent, and the sphincters are rarely affected.

Diagnosis.—As an independent affection lateral sclerosis is rare, spastic paralysis of the legs usually being due to (1) *diseases of the brain involving both motor tracts,* such as infantile cerebral palsy, or (2) *diseases of the spinal cord dividing the lateral columns,* such as multiple sclerosis, syringomyelia, tumors of the cord, compression in Pott's disease, transverse myelitis, etc.

Prognosis.—The disease is incurable, but the course is extremely slow.

Treatment.—Rest, warm baths (90° F.), and massage are the most useful measures. If there be a suspicion of syphilis, antiluetic treatment should be instituted.

AMYOTROPHIC LATERAL SCLEROSIS

Definition.—A chronic disease, characterized anatomically by degeneration of the anterolateral columns and atrophy of the ganglionic cells in the anterior gray horns of the spinal cord, and clinically by loss of power, atrophy, and a spastic state of the muscles.

Pathology.—The chief lesion is a degeneration of the pyramidal tracts, with atrophy of the large cells in the ventral horns and of certain groups of cells in the medulla.

Symptoms.—These include wasting of the muscles, with loss of power, spastic contractions, and exaggerated reflexes. The upper extremities are usually first affected.

When the medulla is involved, symptoms of glossolabial laryngeal paralysis appear. Sensation is not impaired, and the sphincters are rarely disturbed.

The muscular rigidity and exaggerated reflexes will distinguish it from pure *progressive muscular atrophy,* and the atrophy of the muscles from pure *lateral sclerosis.*

Prognosis and Treatment.—The course of the disease is exceedingly slow, but invariably progressive. The treatment is that of primary spastic paraplegia.

BULBAR PARALYSIS

(Glossolabiolaryngeal Paralysis)

Definition.—Paralysis of the lips, tongue, pharynx, and larynx from degeneration of the motor nuclei of the medulla oblongata.

Etiology.—An acute form is observed that results either from hemorrhage or from an acute poliomyelitis of the medulla. The chronic form (*progressive bulbar paralysis*) is essentially a chronic poliomyelitis of the bulb. It may occur as an independent disease, but more often it is a part of amyotrophic lateral sclerosis or progressive muscular atrophy.

Symptoms.—These include impairment of speech; inability to protrude the tongue; dribbling of saliva; difficult swallowing; choking spells from the entrance of food or mucus into the larynx; partial suppression of the voice with measured speaking; and a lack of facial expression. The paresis is attended by atrophy and fibrillary tremors.

In the rare disease known as *pseudobulbar paralysis,* which results from bilateral lesions in the motor cortex or internal capsule, there is usually some mental impairment and aphasia, and the paralysis is not accompanied by atrophy and fibrillary tremors. *Asthenic bulbar paralysis* (*myasthenia gravis*) is distinguished by the extreme fatigue after exertion, the ready exhaustibility of the muscles with faradism, the involvement of the ocular muscles and limbs, and the absence of atrophy.

Prognosis.—The outlook is unfavorable. The acute variety is speedily fatal; the chronic form may last several years. Death may result from exhaustion, cardiac failure, or aspiration-pneumonia.

Treatment.—This is unsatisfactory. Massage and electricity may be tried. Strychnin has been recommended. The stomach-tube should be used when the patient is unable to swallow.

ACUTE ASCENDING PARALYSIS

(Landry's Disease)

Definition.—An acute disease of rare occurrence, characterized by motor paralysis beginning in the feet and rapidly spreading until it involves the muscles of respiration and deglutition.

Etiology.—The causes are unknown. It is usually observed in young male adults. The disease usually occurs between the ages of twenty and thirty years, and in many cases is probably acute poliomyelitis of the adult or a form of acute myelitis.

Pathology.—In some cases degenerative changes have been found in the ventral horns.

Symptoms.—Febrile symptoms usually usher in the attack. The paralysis begins in the legs and involves successively the trunk, upper extremities, and muscles of respiration and deglutition. The reflexes are abolished. The sphincters are retentive; sensation is usually normal, but there may be some paresthesia; the muscles are relaxed, but do not waste or yield the reactions of degeneration. In some instances the spleen and lymphatic glands are swollen.

Diagnosis.—*Acute Myelitis.*—Anesthesia, wasting, reactions of degeneration, and early involvement of the sphincters will serve to distinguish myelitis from acute ascending paralysis.

Multiple neuritis can usually be distinguished from Landry's disease by the marked sensory disturbances in the former.

Prognosis.—The majority of cases terminate fatally in from a few days to two or three weeks. Occasionally the disease comes to a standstill and a slow recovery ensues.

Treatment.—The patient should be kept at rest, and wet cups applied to the spine. Ergotin (10 to 20 grains a day), belladonna, salicylates, mercury, and iodids are the remedies that have been recommended.

LOCOMOTOR ATAXIA

(Tabes Dorsalis; Posterior Spinal Sclerosis)

Definition.—A degenerative affection of the spinal sensory neurones, often involving also the sensory neurones of the cranial nerves, and characterized by incoördination, loss of deep reflexes, disturbances of sensation and nutrition and various ocular phenomena.

Etiology.—The disease occurs most frequently between the ages of thirty and fifty. It is ten times more common in men than in women. Syphilis is the chief, if not the only, exciting cause. Exposure, excesses, overexertion, and alcoholism are contributing factors.

Pathology.—The pia mater over the posterior columns is somewhat thickened and opaque. The posterior columns have a grayish color and are firm and shrunken.

Microscopic examination reveals degeneration and sclerosis of the dorsal-roots (usually in the lumbar region) and of the tracts and columns which these roots form in the spinal cord—tract of Lissauer, columns of Goll, Burdach and Clarke. The fibers involved are concerned in conveying impulses of tactile sensibility, deep sensibility, especially muscular and [illegible]

legs, and frequently regarded as "
of paresthesia such as numbness a
a sense of constriction about the
ances of the urinary and sexual fun
type; loss of the deep reflexes, e
(Westphal's sign); reflex iridoplegi
the pupil showing no reaction to li
in convergence and accommodatio
called visceral crises, the most fam
crisis, characterized by paroxysms
incessant vomiting, and the secretio
isolated areas of hypesthesia or
the root or segmental type, about th
ties; and an excess of lymphocytes,
and a positive Wassermann reactio

In many cases accompanying t
there are other ocular disturbances,
transient or persistent paresis of th
manifested by squint, ptosis or do
dimness of vision, the result of optic

One of the first obtrusive sympt
want of certainty and precision in

steps are awkward and jerky, the foot is raised high, projected forward and outward and brought down forcibly with a thud, the body is bent forward, and the eyes are riveted to the floor. Although there is no loss of muscular power in this stage, the muscles are abnormally flaccid and in consequence passive movements of unusual extent are possible (hypotonia). In many cases trophic phenomena also ensue, especially a perforating ulcer of the sole of the foot, abnormal brittleness of the bones, and the so-called arthropathies or Charcot's joints—painless swellings of the large joints, with effusion, atrophy of the bones and cartilages, and ultimately luxation.

In from five to twenty years or more if the patient does not die of some intercurrent disease, the *paralytic stage* supervenes. This is characterized by progressive muscular weakness, incontinence of urine and feces, cystitis, bed-sores, and increasing marasmus.

In a certain proportion of cases the symptoms of paretic dementia (see p. 455) are associated with those of locomotor ataxia, giving rise to the condition known as *tabo-paralysis*.

Diagnosis.—*Cerebellar disease* may be differentiated by the form of the ataxia (reeling, with a tendency to fall in a certain direction), headache, vomiting, double optic neuritis, and absence of pupillary rigidity, Romberg's sign, Westphal's sign, shooting pains and spinal lymphocytosis. *Multiple neuritis* differs from tabes in the early appearance of motor paralysis, muscular atrophy, and tender nerve trunks, and in the absence of pupillary rigidity, lightning pains, girdle sensation and vesical disturbances.

Prognosis.—Complete recovery probably never occurs. The duration ranges from three to twenty years. Death is usually the result of some intercurrent disease.

Treatment.—Rest is an important factor in the treatment. Erb advises that the patient should live as if he were an old man, quietly, regularly, and with no excitement. Mental fatigue should also be avoided. Sexual excesses are exceedingly injurious. The diet should be nutritious and easily digestible. Alcohol and tobacco should be used sparingly, if at all. Flannel should always be worn next to the skin.

Massage affords a valuable means of securing the benefits of exercise without the expenditure of energy.

Systematic reëducation of coördinating movements, as originally recommended by Frenkel, has been found the most effective remedy for the ataxia. Even in advanced cases, in which there is marked disturbance of sensation, this method of treatment is not without benefit, and the improvement may last for years if the disease is stationary or only slowly progressive.

Tepid baths of 80°–85° F. are sometimes of distinct service. They should be suspended, however, while the exercise treatment is being used. Salvarsan, mercury and iodids should be given a thorough trial in all cases in which syphilis is suspected.

The Pains.—When the pains are severe, the most potent remedial measure is absolute rest in bed. Light touches of the actual cautery or sinapisms over the root of the nerve supplying the affected part often afford relief. Deep massage is sometimes of service. Mitchell has found the alternate application of ice and hot water useful. Flannel bandages applied firmly from the toes up to the middle third of the thigh sometimes do much good. A snugly fitting abdominal binder may also be used to lessen girdle pain. Electricity in the form of the faradic brush, static spark, or stabile galvanic anode is worthy of a trial. Division of the posterior nerve-roots has recently been recommended.

The most generally useful anodynes are phenacetin and antipyrin. Nitroglycerin occasionally succeeds. In many cases recourse must be had to morphin, but its use should be deferred as long as possible.

Numbness and *paresthesia* often yield for a time to local applications of faradism given with the wire brush.

Vesical weakness should receive the most careful attention. The bladder must be completely emptied—if need be, by catheterization. On the first appearance of cystitis the bladder temporal sides of the optic discs; and (6) various subjective disturbances of sensation, such as headache, giddiness, and numbness or tingling in the limbs. In addition there may be diplopia, paresis of the ocular muscles, disturbances of micturition, mental impairment, hypesthesia, and epileptiform or apoplectiform seizures. The course is long, from two to twenty years, and remissions not rarely occur.

Diagnosis.—*Paralysis Agitans.*—In this disease the face is mask-like and the attitude and gait are characteristic. The tremor decreases during effort and persists during repose; nystagmus and scanning speech are absent. *Hysteria* may simulate multiple sclerosis, but it does not present nystagmus, optic atrophy, or bladder disturbances.

Treatment.—The general treatment is the same as that of posterior sclerosis. Bromids, hyoscin, hyoscyamin, and belladonna have been recommended for the tremors.

HEREDITARY ATAXIA

(Friedreich's Ataxia)

Definition.—A family disease, characterized anatomically by sclerosis of the lateral and posterior columns of the spinal cord, and clinically by symptoms resembling those of locomotor ataxia.

Etiology.—The disease most frequently develops between the second and fifteenth years, and usually attacks a number of individuals in the same family. Some cases can be traced to heredity; in others no cause can be ascertained.

Pathology.—The chief lesion is a sclerosis of the lateral and posterior columns of the spinal cord.

Symptoms.—The characteristic symptoms are: Ataxia of all purposeful movements; loss of the knee-jerks; irregular jerking movements of the hands; muscular weakness; nystagmus; a scanning speech; lateral spinal curvature, and deformity of the feet, generally talipes equinus with extension of the big toe.

It differs from *locomotor ataxia* in the absence of sharp pains, of anesthesia, of the Argyll-Robertson pupil, and in the occurrence of nystagmus, scanning speech, and deformities. *Hereditary or family cerebellar ataxia* (*Marie's ataxia*) clinically resembles Friedreich's ataxia, but differs from it in its later onset, normal or exaggerated knee-jerks, and absence of deformities.

Course.—The disease is slowly progressive, and treatment is of no avail.

SYRINGOMYELIA

Definition.—A chronic affection of the spinal cord, characterized anatomically by the formation of a cavity in its substance, and clinically by atrophy of certain muscles, peculiar disturbances of sensation, and various trophic disorders.

Etiology.—It is much more common in males than in females. Eighty per cent. of the cases occur between the ages of ten and forty years. Acute infective diseases and traumatism are mentioned as causes.

Pathology.—The cavity is probably produced in the majority of cases by softening of overgrown neuroglia tissue (gliosis), which exists in consequence of some developmental anomaly. The cervical and upper dorsal regions are the usual seats of the lesion. The cavity lies in the gray matter, and may be in the position of the central canal or somewhat posterior to it. Secondary degenerations are frequently observed in the anterior or posterior horns or in the anterior or posterior columns.

Symptoms.—The disease usually attacks the upper extremities, the chief symptoms being wasting of the muscles; fibrillary tremors; loss of painful and thermic sensations, while tactile sensation is preserved or but slightly affected (*dissociation symptom*); lateral spinal curvature; and various trophic disturbances, such as arthropathies, fissures, ulcers, and gangrene. Such eye symptoms as nystagmus, inequality of pupils, and narrowing of the visual fields are frequently observed. In many instances symptoms of lateral sclerosis, posterior sclerosis, or bulbar disease are superadded.

The distinctive features of *Morvan's disease* (probably a form of syringomyelia) are tactile anesthesia and painless felons.

Diagnosis.—*Cervical pachymeningitis* is more painful, and the anesthesia includes tactile sensation. In *progressive muscular atrophy* and *amyotrophic lateral sclerosis* sensory symptoms are wanting. *Leprosy* may be recognized by loss of tactile sensation, discoloration of skin, nodular swellings, and presence of bacilli in the tissues.

Course and Treatment.—The course is slow, extending over many years, and the treatment is purely symptomatic.

CAISSON DISEASE

(Divers' Paralysis)

Definition.—A condition observed in divers and others subjected to increased atmospheric pressure, and characterized by motor and sensory paralysis and other nervous symptoms.

Etiology.—A pressure of more than two atmospheres is usually required to produce the paralysis, and the time elapsing before its appearance lessens as the pressure increases.

Pathology.—The symptoms are apparently due to congestion, and foci of hemorrhages and softening in the cord caused by the liberation of gases (chiefly nitrogen) that have been absorbed during exposure to the high pressure.

Symptoms.—The condition may manifest itself immediately on reaching the surface or after the lapse of several hours. The most important phenomena are pains in the joints followed by motor and sensory paralysis in the lower extremities. The bladder and rectum are sometimes involved. Occasionally the paralysis takes the form of a hemiplegia or monoplegia instead of a paraplegia. Gastralgia and vomiting are common symptoms. In severe cases coma develops and death follows in a few hours. The paralysis may be permanent, but, as a rule, recovery ensues within a few days or weeks.

Treatment.—As a preventive measure, the transition from high to low pressure should be accomplished gradually. If changes, the prognosis should

Treatment.—The cause s possible, removed. The par the pain, sedative lotions (le fomentations, or small blisters parts, and salicylic compoun ministered. Such a combina efficacious:

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SIG.—One every three or fo

After the acute symptoms hav and strychnin (preferably hypo to promote nutrition.

MULTIPLE

Definition.—Inflammation c ing from a general cause, and ch anesthesia, paresis, and muscul

Etiology.—The disease may

Symptoms.—The **acute form** is characterized by chilliness, moderate fever (102°–103° F.), pains in the head and back, anorexia, constipation, and the following local phenomena: pain, numbness, and tingling in the affected limbs, loss of power, especially in the legs and extensor muscles of the wrist, tenderness over the nerve-trunks, abolition of reflexes, and, later, hypesthesia or anesthesia.

Death may occur within one or two weeks from cardiac or respiratory paralysis. Usually, however, recovery follows in from a few weeks to several months.

Chronic Form.—*Sensory symptoms* are usually prominent. Numbness, tingling, hyperesthesia, and intermittent pains appear early and are followed by hypesthesia or anesthesia, especially of the legs and hands. The nerve-trunks are sensitive. In certain forms of neuritis, as that resulting from diphtheria or from lead poisoning, sensory disturbances are frequently absent.

Motor Symptoms.—Weakness of the legs and forearms develops rapidly. The typical paresis is foot-drop and wrist-drop. The sphincters are not affected. The knee-jerks are lost. As a rule the paralyzed muscles do not respond to the faradic current, but yield the reactions of degeneration with the galvanic current. If the patient is able to walk, his gait is characteristic. To avoid dragging his toes, he raises the foot high, throws it suddenly forward, and brings it down flat on the floor, as if walking over obstacles (steppage gait).

Vasomotor and Trophic Disturbances.—The paralyzed muscles are flabby and soon waste. Edema of the feet and hands, and local sweating are often seen. Changes in the nails are common. Bedsores do not appear.

Mental Symptoms.—Forgetfulness, confusion, disorientation, and narration of pseudoreminiscences (Korsakow's syndrome) are not rare, especially in the alcoholic form.

Diagnosis.—*Locomotor Ataxia.*—The absence of the lightning-pains, girdle sensation, Argyll-Robertson pupil, and the presence of paralysis, wasting, and neural tenderness will serve to distinguish multiple neuritis from locomotor ataxia.

Prognosis and Treatment.—As a rule, the prognosis is good, if the exciting cause can be removed and the general health of the patient is not seriously impaired. The treatment is similar to that of localized neuritis.

SCIATICA

Definition.—Pain along the sciatic nerve, usually resulting from neuritis.

Etiology.—It may be due to (1) exposure to cold; (2) trauma; (3) infectious diseases; (4) chronic intoxications (gout, alcoholism, diabetes); (5) pressure of an intrapelvic growth, as a uterine fibroid or a carcinoma of the prostate; (6) relaxation or chronic inflammation of the sacroiliac joint.

Symptoms.—The disease may begin abruptly or gradually, and is characterized by a sharp shooting pain running down the back of the thigh. Movement of the limb intensifies the suffering. The pain may be uniformly distributed along the course of the nerve, but not infrequently there are certain spots where it is more intense. Subjective sensations, such as tingling and numbness, are often noted. The nerve may be extremely sensitive to touch. The symptoms grow worse at night and on the approach of stormy weather. In long-standing cases there may be much wasting of the muscles.

Diagnosis.—*Coxalgia.*—In this affection the pain is most marked in the hip- and the knee-joints; pressure over the trochanter elicits pain; and the nerve is not tender to the touch.

Prognosis.—Recovery follows in the majority of cases if treatment is instituted early and is persistently carried out. Relapses, however, are common, and in some cases the pain becomes more or less continuous.

Treatment.—The first indication is to remove the cause. In acute cases rest in bed is essential. In severe cases the limb should be immobilized by means of salt bags or a long straight splint. Free evacuation of the bowels should be secured in order to deplete the pelvic veins. If there is a history of rheumatism or the attack has been induced by cold, salicylates should be given in full doses. If there is reason to suspect syphilis, iodids should be given a fair trial. Irrespective of the cause, phenacetin or antipyrin may be useful in relieving pain. Counterirritation often affords much relief. If the pain is very severe, this is best accomplished by means of small blisters or light touches of the actual cautery applied over the points of greatest tenderness. In some cases acupuncture acts very satisfactorily. In milder cases the Scottish douche—in which a stream of warm water of gradually increasing temperature is directed on the course of the nerve until the pain subsides, when it is suddenly changed for a cold jet—is an efficient remedy. Agonizing pain must be relieved by injections of cocain (¼ grain), quinin and urea chlorid (5 grains), chloroform (5 to 10 minims), or guaiacol (2 to 3 minims), made deeply and as near to the nerve as possible. Morphin should be withheld as long as possible. Massage is indicated only when the acute symptoms have subsided, and should then not be too energetic. In chronic cases due to relaxation of the sacroiliac joint the application of a pelvic brace often gives great relief. In some very obstinate cases exposure of the nerve and the breaking up of adhesions has resulted in a cure.

FACIAL PARALYSIS

(Bell's Palsy)

Etiology.—Paralysis of one side of the face may result: (1) From a tumor, clot, or abscess involving the facial center on the cortex of the brain or the nucleus of the facial nerve; (2) from the pressure of inflammatory exudate on the nerve-trunk between the brain and the skull; (3) from paralysis of the nerve within the petrous portion of the temporal bone, excited by a fracture or by an extension of inflammation of the middle ear; (4) from inflammation of the peripheral filaments, excited by exposure, injury, rheumatism, or one of the infectious fevers.

nerve is involved in the te
taste in the anterior part o

Diagnosis.—When the
muscles of the face (orbi
usually escape, voluntary
emotional movements, elec
is usually hemiplegia.

When the lesion is nucl
of one side of the face, in
eye, are involved, both em
are lost, and the electric
In nuclear lesions other cr
with the facial. In pontin
of the limbs on the side op
paralysis). When the nerv
canal there is frequently lo
the tongue on the paralyzed

Prognosis.—The prognos
should be guardedly favor
peripheral neuritis.

Treatment.—The cause
possible, removed. In

FUNCTIONAL NERVOUS DISEASES

HEADACHE

(Cephalalgia)

Headache of Organic Brain Disease.—This form is observed in meningitis, intracranial syphilis, cerebral tumor, abscess, softening, etc., and is usually distinguishable by its persistence and association with other evidences of organic cerebral disease, such as vertigo, vomiting, optic neuritis, and so-called focal symptoms. Headache due to intracranial syphilis is usually worse at night, and associated with somnolence and other signs of syphilis, such as a positive Wassermann reaction, etc.

Headache of Cerebral Hyperemia and Cerebral Anemia.—*Active cerebral congestion* usually results from prolonged mental work, fever, or exposure to the sun. Toxic and reflex headaches are often directly due to active cerebral congestion, but these will be discussed later.

Passive cerebral congestion may result from obstruction to the return of blood from the brain, as by a tumor of the neck or cardiac disease. It is also common in elderly persons from a relaxed condition of the vessels.

In cerebral congestion the headache is usually diffuse and of a throbbing character; the head is often hot and the face flushed.

Cerebral Anemia.—This is frequently dependent upon general anemia. It is also common in neurasthenia resulting from overwork, prolonged emotional excitement, excesses, etc. More rarely it is dependent upon aortic stenosis. The pain is frequently vertical; it is not throbbing, but it is described as a sensation of weight or gnawing; the extremities are cold; the face and eye-grounds are pale; the mind is depressed; fainting spells are sometimes present, and lowering the head relieves the pain.

The exciting cause of cerebral hyperemia or anemia must be determined by the clinical history and a thorough examination of the various organs and blood.

Reflex Headache.—Headache is often due to *eye-strain*, and in obstinate cases a careful examination of the eyes should always be made. Headache of this origin is frequently a browache, and may be associated with restlessness, vomiting, and insomnia. It is induced or aggravated by prolonged use of the eyes.

Ovarian or uterine diseases often occasion a reflex headache. It is usually located at the vertex, and is relieved by pressure of the hand.

Gastric irritation is responsible for some headaches; the latter are relieved by vomiting, and are usually associated with other evidences of stomachic disorder.

Nasal catarrh may induce persistent headache, which is usually confined to the forehead, temples, or vertex, and is aggravated by exacerbations of the catarrh. The pain is associated with tenderness of the inner wall of the orbit, and is increased by irritating the nasal mucous membrane with a probe.

Toxemic Headache.—A persistent headache often results from Bright's disease, and is *uremic* in origin. It may be recognized by the high arterial tension and by the albumin and casts in the urine. A urinary analysis should be made in all cases of persistent headache.

Among other headaches of toxic origin may be mentioned the headaches due to alcoholism, to excessive use of tobacco, tea or coffee, to gout, to diabetes, to the acute infections.

Hysteric and Neurasthenic Headache.—In *hysteria* there is frequently a persistent headache, which grows worse at the menstrual periods, and which improves under pleasurable excitement. It may be diffuse, but frequently it is localized, and is described as resembling the effect that would be produced by a nail being driven into the head; hence it has been termed *clavus*.

The headache of *neurasthenia* is, as a rule, a dull pain or merely a sensation of fatigue. It is usually occipital but it may be diffuse. It is almost invariably aggravated by mental or physical effort and is associated with other neurasthenic phenomena such as ready fatigue, backache and sleep disturbances.

Indurative Headache.—This comparatively rare form of headache, sometimes referred to as "rheumatic," is frequently excited by chilling. It is increased by movements affecting the muscles of the head, and is associated with tenderness of the scalp and the presence of sensitive nodular swellings at points upon the skull corresponding to the insertion of the muscles.

Diagnosis.—Headache must be distinguished from *migraine*. In the latter the attacks are usually more distinctly periodic; the pain is often unilateral, and is frequently accompanied by vomiting, vasomotor disturbances, and subjective visual phenomena.

Headache in the region of the orbit may be mistaken for *acute glaucoma*, but in the latter condition the eye is inflamed; the cornea is hazy; the pupil is sluggish; vision is impaired; and on palpation the affected eyeball is found to be harder than its fellow.

Treatment.—In the interval between the attacks careful search should be made for the cause, which, if possible, must be removed. In the reflex headache of eye-strain the adjustment of proper glasses is often all that is required. In the headache of gastric origin appropriate remedies should be directed to the stomach. In the headache of anemia a nutritious diet, with iron, arsenic, and other tonics, will be required. In headaches of uremic origin a milk diet with measures calculated to increase the action of the skin, bowels, and kidneys will

℞. Sodii bromidi . . .
Spiritus ammoniæ ar
Aquæ . . . . . .
Sig.—A tablespoonful eve

In headache of acute cere
be soaked for ten or fifteen
ice-bag placed on the head;
lowing administered:

℞. Acetphenetidini. . .
Sodii bromidi. . . .
Fiant chartulæ No. xij.
Sig.—One powder every ho

When the attack is very sev
be given every hour or two.

In cerebral anemia relief t
antipyrin or phenacetin, especia
thus:

℞. Acetphenetidini. . .
Caffeinæ citratæ . .
Fiant chartulæ No. xij.
Sig.—One as required.

In indurative headache salic

draft, and diuresis encouraged by digitalis, caffein, or the vegetable salts of potassium.

VERTIGO

(Dizziness; Giddiness; Swimming in the Head)

Definition.—A sense of unstable equilibrium in which the patient himself or surrounding objects appear to be in a state of rapid oscillation or rotation. It is a symptom of many conditions.

Etiology.—Vertigo may result from:

1. Disturbances of the cerebral circulation. The vertigo occurring in arteriosclerosis, fatty heart, valvular heart disease, and the anemias is included under this head.

2. Organic disease of the brain. Vertigo is especially common in lesions of the cerebellum, but it may also be present in lesions of the cerebrum.

3. Neuropathic conditions. Vertigo is not uncommon in hysteria, neurasthenia, traumatic neuroses, migraine, and epilepsy. In epilepsy it may precede, follow, or take the place of a convulsion.

4. Aural disturbances. Vertigo is most frequently observed in lesions of the labyrinth (Ménière's disease), but it may make its appearance as a result of disease of the middle ear.

5. Ocular disturbances. Ocular vertigo is usually dependent upon paresis of the ocular muscles, and is probably due to false projection of the retinal images. It is relieved by closing the eyes.

6. Toxemic conditions. Vertigo is sometimes observed in indigestion, gout, uremia, diabetes, acute infections, and in poisoning by tobacco, alcohol, lead, and many other substances.

7. Mechanical causes. The vertigo experienced in seasickness, swinging, whirling, etc., is probably dependent upon violent excitation of the semicircular canals, produced by the rapid movements of the body.

duration of the attacks
usually serve to differen
The treatment of vertig
the affection of which it

MÉNIÈ

The term Ménière's disea
characterized by deafness,
of intense vertigo. The pa
at intervals of weeks, may
syncope. The condition
but occasionally recovery e
the affected side becomes a
in the labyrinth and may b
hemorrhage. In some ins
middle ear. It should be
cerebello-pontile angle may

Treatment.—The middle
and any existing disease t
the ear (actual cautery or s
mids in full doses, gelsemiu
thrice daily) may be of se
(Ch

Etiology.—A neuropathic inheritance is the most important tiologic factor. Chronic alcoholism and syphilis in the arents may also act as predisposing causes. Not rarely he disease follows an injury of the brain or one of the nfectious fevers. The reflex convulsions of infancy, if fre- uently repeated, may serve as an exciting cause in those pre- lisposed. Essential epilepsy usually begins before the age f puberty, and rarely develops after the thirtieth year. In pileptic subjects overwork, digestive disturbance, excitement, r menstruation may be the immediate cause of an attack.

Pathology.—No constant lesions are revealed at autopsy. Iorphologic anomalies in the cerebral convolutions and in the rganism as a whole indicative of defective development are, owever, frequently noted. The disease depends apparently pon an excessive irritability of the motor cells of the cerebral ortex.

Symptoms.—*Grand Mal.*—In about one-half of the cases he convulsion is preceded by a warning symptom, or aura. This may consist of a sensation of numbness beginning in ne extremity, muscular twitchings, flashes of light, a peculiar dor, or a certain emotion. After the aura the patient emits shrill cry, becomes pale, and falls to the floor unconscious nd convulsed. At first the body is thrown into a tonic spasm, vith the head rotated and retracted, the limbs forcibly ex- ended, and the thumbs turned into the palms and firmly lenched by the fingers. The respiration ceases and cyanosis ollows. In a few seconds the tonic spasm gives way to a clonic pasm in which the movements are jerky, rapid, and often iolent. During this stage the breathing is stertorous, the ongue is often bitten, frothy saliva, frequently blood-streaked, xudes from the mouth, and the urine is discharged. The whole ttack usually lasts two or three minutes. At the close, the atient may remain in coma for a variable time or he may apidly recover consciousness, and afterward complain of nental confusion, headache and muscular soreness. Occasion- lly the attack is followed by a trance-like state, by epileptic

denly stops in the midst
still for a few seconds,
tivity, as if nothing had
runner of *grand mal* or m

Between the extremes
of every degree of severi
occur daily, several time
months, or years.

The term *status epilep*
which convulsions follow
without a return of conscio

No other symptoms may
pertaining to the paroxysm
frequently recur mental
supervenes.

Diagnosis.—In *organic*
symptoms are usually pres
are limited to one member
and then become general
thalmoscopic examination
vulsions occurring for the
years of age are usually

Treatment.—Hygienic treatment is of the utmost importance. Moderate exercise, both mental and physical, is beneficial. Idleness and seclusion have a baneful effect. Home training must be carried on with the greatest care, much tact and firmness being required to prevent loss of self-control. Children who are particularly irascible are often much better trained in a special institution. As a rule, a diet that is for the most part vegetable will be found to be best adapted to the patient's condition, but when the disease is associated with lowered vitality, a fair amount of animal food should be permitted. Overloading the stomach is a potent factor in precipitating attacks. The bowels must be regulated by diet, and, if necessary, by mild aperients. Liberal water-drinking, frequent bathing, followed by friction of the skin, light exercise in the open air, and other measures that favor elimination are to be recommended. General tonics, like iron, arsenic, and cod-liver oil, are sometimes required to combat anemia and malnutrition.

Although very few cases of epilepsy are purely reflex, local irritation—phimosis, adherent prepuce, worms, a foreign body in the nose or ear, and painful cicatrices—should be carefully sought for and, if found, removed.

The most reliable drugs are the bromids: one or two drams of a combination of the bromids of sodium, potassium, and ammonium may be given daily. Strontium bromid is often efficacious, and it is less depressing than the other bromids. The tendency to acne may be lessened by the addition of a drop or two of Fowler's solution to each dose of bromid. A small amount of antipyrin sometimes lessens the amount of the bromid required to check the convulsions.

℞. Potassii bromidi	
Ammonii bromidi	āā ʒiij
Liquoris potassii arsenitis	fʒss
Antipyrinæ	ʒj
Aquæ menthæ piperitæ	q. s. ad f℥vj.—M.

SIG.—A tablespoonful in water night and morning.

cent. of recoveries.

The Attack.—If there
may be averted at times
Patients may provide the
pearls that may be crushe
aura is felt in a limb, the
circled with a light ligat
should be slightly raised,
of cork, wrapped in a
teeth. In the status epil
are inhalations of chlorofo
dermic injections of hyosc
grain), enemas of chloral (

H

Definition.—A psychon
normal susceptibility to su
and manifested by a trai
character.

Etiology.—Females are
occasionally develops in m

Freud ascribes hysteria to a mental conflict (suppression of memories) which has arisen as a result of painful experiences (often sexual events) in early childhood, and would make the vivid recollection of these experiences, under insistent questioning, and a complete verbal confession of them to the physician, an essential element of treatment.

Symptoms.—The various manifestations may be described under three heads: (1) Motor, (2) sensory, and (3) psychic.

Motor Phenomena.—Paralysis not infrequently results from hysteria; it may take the form of a hemiplegia, paraplegia, or monoplegia, although the first is by far the most common. The paralysis is usually paroxysmal, and is frequently associated with contractures and anesthesia. The affected muscles do not waste.

Local paralysis is also common; thus there may be aphonia from paralysis of the vocal cords; dysphagia, from paralysis of the esophagus; and incontinence of urine, from paralysis of the bladder.

Convulsive seizures are common manifestations of hysteria, and may closely simulate the paroxysms of true epilepsy, but there is no aura; the patient usually falls in a comfortable place; consciousness is only apparently lost; the tongue is rarely bitten; the eyes are partially closed; the face is expressive of some emotion; screaming or sobbing is of frequent occurrence; the movements are usually tonic, or if clonic, of wide range and purposive; the seizures are of long duration, and may be continued for several hours, and there is no involuntary micturition.

The spasms may be local; thus there may be retention of urine from spasm of the bladder; hiccup, from spasm of the diaphragm; dysphagia, from spasm of the esophagus; or a "phantom tumor," from spasm of the abdominal muscles.

Among other motor phenomena may be mentioned obstinate tremors, choreiform movements, and muscular contractures.

Sensory [illegible] —There may be a complete loss of sensation [illegible] ts, as of one side of the body.

thesia without other nervous phenomena is usually hysteric. In some cases tactile sensation is preserved and there is a loss only of thermic or painful sensations. The anesthetic part is sometimes unusually pale, and when pricked with a needle, may fail to bleed.

The special senses may be involved; thus there may be contraction of the field of vision, complete blindness, loss of smell, loss of taste, or loss of hearing. These palsies are usually transient, and sometimes alternate with one another.

Instead of anesthesia, there may be hyperesthesia or pain. An intensely painful and tender condition of the abdomen may be mistaken for peritonitis. A localized pain in the head, described as resembling the effect of a nail being driven into it, is termed hysteric *clavus*. The joints occasionally become swollen and tender, resembling arthritis (neuromimesis).

Psychical Phenomena.—As a rule, the most conspicuous psychic phenomena are an abnormal susceptibility to suggestion, introspectiveness, incapacity to exercise control over emotions and actions, and a morbid craving for sympathy and attention. Hysterical patients are excitable, highly mercurial, and easily moved to laughter or tears. Occasionally delirium, ecstasy, catalepsy, trance or somnambulism makes its appearance.

Treatment.—This must be directed both to the mind and the body, especially to the former. To be successful, the physician must be able to inspire absolute confidence and faith in the mind of the patient. She must be impressed repeatedly with the fact that her condition is a curable one, and that with her thorough coöperation restoration to health will certainly follow. To intimate that her symptoms are feigned or are wholly within her control is a grave error. In many cases no method of treatment proves successful until the patient has been removed from her customary surroundings and separated from her sympathetic relatives and friends.

Suggestion is employed consciously or unconsciously in the treatment of hysteria by every successful physician. Without it many of the remedies recognized as efficacious become wholly impotent. Complete hypnotism, however, is by no means so generally useful as continuous suggestion, and, moreover, in the event of failure, seems capable of still further lowering the will-power and of increasing the emotional excitability.

The physical condition of the patient must not be neglected. General measures, such as hydrotherapy, systematic exercise in the open air, massage, and electricity, are valuable aids to recovery. In grave cases the treatment associated with the name of S. Weir Mitchell often yields admirable results. It consists in isolation from sympathizing friends and relatives; abundant feeding, especially with milk; and complete rest of body and mind, with passive exercise obtained by massage and electricity.

Except to meet underlying conditions and to combat special symptoms, drugs are of little value. Iron and arsenic are useful when there is anemia. Antispasmodics, like valerian, sumbul, asafetida, and camphor, are serviceable in allaying abnormal nervous irritability.

Such combinations as the following may prove useful:

℞.	Arseni trioxidi		gr. ss
	Ferri pyrophosphatis		
	Extracti sumbul	āā	gr. xx
	Asafœtidæ		gr. xl.—M.

Fiant pilulæ No. xx.
SIG.—One three or four times a day.

℞.	Quininæ valeratis		
	Zinci valeratis		
	Ferri valeratis	āā	gr. xxiv.—M.

Fiant pilulæ No. xxiv.
SIG.—One pill thrice daily.

Occasionally more powerful sedatives, like the bromids, phenacetin, and chloralamid, may be demanded, but the continuous use of such remedies is always to be condemned. Such drugs as morphin, alcohol, and chloral are distinctly dangerous.

When hysteria is complicated by local disease, special treatment will be required, but no operation should ever be performed for the relief of nervous symptoms unless there exists an actual organic lesion.

Convulsions.—Isolation of the patient is imperative. Firm pressure over the ovarian region is sometimes successful. The affusion of cold water over the face is useful. Inhalations of amyl nitrite, or even of chloroform, may be employed if necessary. Strong faradic currents applied to the spine are occasionally efficacious.

Anesthesia is best treated by electricity, especially by the faradic brush. Static electricity, owing to the profound mental impression which it produces, is also useful. *Hyperesthesia* and *pain* often yield to the continuous or interrupted galvanic current. In *paralysis* the patient should be instructed how to regain, by long-continued practice, the use of the affected part. Swedish movements, massage, and faradization are useful adjuvants. In *aphonia* the faradic current, applied by means of special electrodes, is the most reliable remedy. In *contractures* the most useful measures are massage, passive movements, and faradization.

NEURASTHENIA

(Nervous Prostration)

Definition.—A functional disease characterized by a lack of nervous energy and increased sensitiveness to external impressions.

Etiology.—The predisposing causes are much the same as those that give rise to hysteria. Men are often affected. Overwork, prolonged mental strain, depressing emotions, physical injury, acute infections, sexual excesses, and chronic intoxications are important determining causes.

Symptoms.—*Cerebral Symptoms* (*Psychasthenia*).—These include depression of spirits, inability to concentrate the mind

ne subject for any length of time, insomnia, vertigo, head-
, irritability of temper, introspection, and various forms
morbid fear, as of crowds, closed spaces, darkness, etc.

pinal Symptoms.—The chief manifestations are: Pain in
back, spots of tenderness along the spine, weakness of
extremities, marked prostration after moderate exertion,
various subjective phenomena, such as numbness, ting-
, formication, and neuralgic pains.

astro-intestinal Symptoms.—These consist of anorexia,
ted tongue, indigestion, and constipation.

irculatory Symptoms.—These include palpitation, tachy-
lia, pseudoangina, cold extremities, and sometimes violent
sation of the abdominal aorta.

exual Symptoms.—In females there is often amenorrhea
dysmenorrhea, and in males impotence or spermatorrhea.

iagnosis.—The diagnosis is rarely difficult. Before rele-
ing a case to this class, care must be taken to exclude
nic disease of the various organs and *such disorders* as *gout,*
betes, exophthalmic goiter, and anemia.

ysteria.—This affection may be distinguished by the
upt onset, the intermittent character of the symptoms, and
h stigmata as paralysis, anesthesia, contractures, emotional
breaks, and convulsions.

rognosis.—If the cause can be removed and the patient
trolled, the prognosis is favorable.

reatment.—The treatment is largely hygienic and dietetic,
must vary considerably in different cases. If there has
n inactivity, regulated physical exercise will be of great
ue; on the other hand, the weak and anemic will require
. In the latter case the plan of treatment introduced by
Weir Mitchell, and known as the "rest-cure," often gives
liant results. In all cases careful attention must be given
he diet, bathing, and clothing, and the patient assured that
is suffering from no incurable disease. Frequent bathing
h salt water, followed by friction of the skin, will often add
he general vigor. Tobacco and alcohol must be interdicted,

and tea and coffee used very sparingly. Tonics, like iron, arsenic, quinin, strychnin, and phosphorus, are often indicated.

ACUTE CHOREA

(Sydenham's Chorea; Chorea Minor; St. Vitus's Dance)

Definition.—A nervous affection occurring especially in children, and characterized by involuntary, jerky muscular contractions that increase under excitement and cease during sleep.

Etiology.—The large majority of cases occur in children between the ages of five and fifteen, though adults, especially women during or after pregnancy, are occasionally attacked. More females are affected than males. Heredity sometimes plays an important rôle. Children of a nervous temperament are especially susceptible. Fright or shock is frequently an exciting factor. In about one-fifth of all cases there is an antecedent history of rheumatism between which disease and chorea there appears to be a close relation. Chorea is most common in the spring months.

Pathology.—There are no constant lesions, but hyperemia, minute hemorrhages, emboli and spots of softening in the motor tracts have been found in some cases. These changes are the result of an infection, and most frequently of rheumatism.

Symptoms.—The first manifestations are usually restlessness and awkwardness in movement. The child cannot remain still, but is constantly raising its shoulders, jerking its head, twisting its fingers, or shuffling its feet. Frequently these symptoms develop so insidiously that the disease is not recognized, and the child is punished for being fidgety.

When the disease is fully established, the disorderly movements become more marked. They may be confined to one member or may involve the entire body. If the facial muscles are affected, the most grotesque expressions are produced; involvement of the arms may interfere with eating and dressing; if the legs suffer, the gait becomes jerking and stumbling;

involvement of the larynx causes stammering, and spasm of the muscles of deglutition induces difficult swallowing and choking spells. When the attention is directed to the movements, they invariably grow worse, but they diminish during repose and cease entirely during sleep. Sometimes, in addition to the involuntary movements, there is a distinct loss of power in the affected members. The general health is usually more or less impaired. The child is, as a rule, irritable and mentally dull.

Chorea Insaniens.—This form occurs chiefly in adults and most frequently in pregnancy. The movements are very violent, almost constant, and in many cases associated with delirium and fever. Death frequently results from exhaustion.

Diagnosis.—The recognition of chorea is rarely attended with difficulty. In *habit spasm* or *tic* the movements are coordinated, purposeful, more localized and partly or completely under the patient's will.

Huntingdon's Chorea.—This disease is usually hereditary; it rarely develops before the age of thirty; it runs a chronic course; and it is characterized by slower and more incoördinate movements than occur in acute chorea, by progressive mental failure, and by a marked suicidal tendency.

Other conditions producing choreiform movements have been considered on page 436.

Complications.—Acute endocarditis occurs in from 30 to 40 per cent. of all cases. Pericarditis is occasionally seen. Rheumatic manifestations are sometimes present.

Prognosis.—In simple chorea recovery usually follows in the course of two or three months. Death from heart complications is a rare termination. Relapses are very common. *Chorea insaniens* frequently terminates fatally through exhaustion.

Treatment.—Rest of body and mind is an essential element of the treatment. The child should be taken from school and placed under the most favorable hygienic conditions. Amusement in the open air when the weather is fine is to be recommended. As the child is usually anemic, iron is indicated in the majority of cases. Among the special remedies arsenic holds the first place. Fowler's solution may be given in doses of two drops thrice daily, gradually increased to eight or ten drops thrice daily. If arsenic fails, cimicifuga should be tried. A dose of 10 minims of the fluid extract may be given after meals and gradually increased to ½ fluidram or more. Salicyl compounds and antipyrin are sometimes useful. If the movements are very violent and interfere with sleep, recourse must be had to chloral, bromids, trional, or hyoscin.

NEURALGIA

Definition.—Paroxysmal pain occurring in nerves without obvious anatomical changes.

Etiology.—It is a disease of adults. Women are more frequently affected than men. Heredity is an important etiologic factor. The disease is frequently an expression of anemia. It may result from the action of some toxic agent in the blood; thus it is common in malaria, gout, and chronic lead-poisoning. It may be caused by reflex irritation; thus a trifacial neuralgia may depend on caries of the teeth or eye-strain. In some cases neuralgia results from organic disease of the nerve-centers; thus obstinate trifacial neuralgia may be dependent upon degeneration or tumor of the Gasserian ganglion.

Exposure to cold and wet frequently acts as an exciting cause in susceptible persons.

Symptoms.—The chief symptom is intense pain of a sharp, stabbing character. The area supplied by the affected nerve is usually hyperesthetic, and palpation may detect spots of extreme tenderness where the nerve makes its exit through a bony canal or fibrous sheath—*points douloureux* of Valleix. In some cases the pain is attended with reflex spasm of the muscles. Inspection of the part usually reveals nothing abnormal, but in some cases slight swelling

d. Occasionally herpes precedes or follows an

ck lasts from a few minutes to many hours, and its
may be marked by the passage of a large amount
ne. The interval between the paroxysms varies in
ases; it is frequently several weeks or months. It
rthy that the attacks often recur at regular

Neuralgia (*Tic Douloureux; Prosopalgia*).—In this
pain involves one or more branches of the trifacial
e tender points correspond to the supra-orbital,
l, and mental foramina. Reflex spasms of the mus-
ot infrequently observed. In long-standing cases
the affected side may become coarse and bleached.

al Neuralgia.—In this variety the pain follows the
the intercostal nerves. It is frequently associated
uption of herpes zoster. Spots of tenderness may
d near the vertebral column, in the mid axilla, and
sternum. The possible dependence of intercostal
upon spinal caries or thoracic aneurysm must not
n.

neuralgia involves the upper cervical nerves. A
nderness may be discovered midway between the
rocess and the upper cervical vertebræ. This form
a may also be an expression of spinal caries.

may be a neuralgia, but, as a rule, it is the result of
(see p. 496).

is.—*Neuritis.*—The continuous pain, the tender-
the entire nerve, and the presence of paresthesia,
, paresis, and wasting will serve to distinguish
om neuralgia.

tning-pains of locomotor ataxia must not be mis-
neuralgia. The ataxia, Argyll-Robertson pupil,
sation, bladder disturbances, and absence of knee-
erve to exclude neuralgia.

is.—For the attack the prognosis is good; for per-

are indicated. If there
and iodids should be tri
quinin may effect a cur
expected from regulation
administration of alkalis.
serviceable.

All influences that ten
the nervous system—me
excitement, sexual exc
tea, coffee, or alcohol—sh

In every case an ende
general nutrition, which
measures to be employed
ance of fresh air, proper
tection from the vicissitu
frequent bathing with fric
iron, arsenic, codliver oil,

The following combinat

℞. Quininæ sulphat
Arseni trioxidi .
Ferri reducti . .

peripheral section of the nerve may be employed to advantage. Finally, after failure with all other measures, removal of the Gasserian ganglion or division of the sensory root may be considered. These operations, though not without danger, are usually of lasting value.

The Attack.—Heat, dry or moist, may be applied for its soothing effect. A liniment of aconite or of chloroform is sometimes efficacious. Menthol and chloral camphor are useful in neuralgia of superficial nerves if the pain is slight. Acupuncture and aquapuncture are effective, but are not suitable for use about the face. In obstinate cases active counterirritation by means of blisters or the thermocautery will sometimes be found a potent remedy. In trifacial neuralgia the blisters may be applied behind the ear.

Among the internal remedies most worthy of confidence may be mentioned phenacetin or antipyrin (5 to 10 grains), bromids, cannabis indica, croton chloral, caffein, gelsemium, and salicylic compounds. Morphin is undoubtedly the most certain means we possess of affording temporary relief, but on account of the danger of inducing the opium habit it should be employed only as a last resort. Combinations of a bromid with phenacetin may often be prescribed advantageously. Croton chloral, in doses of from 5 to 10 grains, and tincture of gelsemium, in doses of 10 minims or more, are occasionally serviceable in trifacial neuralgia. Caffein (3 to 5 grains) is sometimes efficacious. Combinations of caffein with phenacetin or with bromids in many cases do more good than single drugs. Neuralgia brought on by exposure to cold and wet is favorably influenced by the salicylates. In such, the following combination will be found of value:

℞. Acetphenetidini
Salophen. . . . . āā ʒiss
Codeinæ sulphatis. . . . . gr. iij.—M.
Fiant chartulæ No. xij.
Sig.—One powder every two or three hours.

MIGRAINE

(Hemicrania; Megrim; Sick-headache)

Definition.—A neurosis characterized by periodic attacks of intense headache, usually unilateral and frequently associated with visual, gastric, and vasomotor disturbances.

Etiology.—It is frequently hereditary. It is more common in women than in men. It usually develops in early life. Anemia, gastric disturbances, gout, eye-strain, menstrual disorders, overwork, and prolonged excitement predispose to it.

Symptoms.—Premonitory symptoms, such as restlessness, depression, and somnolence are common. The attack is often ushered in with visual disturbances, such as flashes of light, colored spectra, dimness of vision, or hemianopsia. The pain is severe and usually limited to the temporofrontal region of one side, though it sometimes spreads until it involves the whole head. The patient is very sensitive to light and sound, and during the attack usually confines herself to a darkened room. Nausea and vomiting frequently supervene. In some cases vasomotor disturbances, such as pallor of the face and coldness of the extremities, or flushing with sweating, are also present.

Less frequent symptoms are numbness or tingling of one extremity, vertigo, tinnitus aurium, and transient motor weakness or aphasia. The duration of the attacks varies from a few hours to several days and of the intervals from a week to several months.

Diagnosis.—*Headache* is less distinctly paroxysmal, less localized, and unaccompanied, as a rule, by visual, gastric, or vasomotor disturbances. In *neuralgia* the pain is limited to the course of a nerve, tender spots are found, and vomiting and visual disturbances do not occur.

Prognosis.—The outlook for cure is not good, but much relief can be afforded, and usually the attacks become less severe or disappear entirely at the menopause or at the age of fifty years.

Treatment.—In the interval the treatment is that of neuralgia. Cannabis indica and arsenic are sometimes of value. Little recommends:

℞. Sodii arsenatis	gr. ij
Extracti cannabis indicæ	gr. vj
Extracti belladonnæ	gr. viij.—M.

Fiant pilulæ No. xxiv.
Sig.—One pill twice daily.

The Attack.—The patient should be kept at rest in a quiet, darkened, well-ventilated room. Antipyrin, phenacetin, caffein, bromids, and salicylic compounds are the most useful remedies. They may often be combined with advantage, as in the following formulas:

℞. Caffeinæ citratæ	gr. xij
Acetphenetidini	ʒj
Sodii bromidi	ʒij.—M.

Fiant chartulæ No. xij.
Sig.—One powder every two hours.

℞. Salophen	ʒiss
Acetphenetidini	ʒj
Caffeinæ citratæ	gr. xij.—M.

Fiant chartulæ No. xij.
Sig.—One every two hours.

PARALYSIS AGITANS

(Parkinson's Disease; Shaking Palsy)

Definition.—A chronic nervous disease, characterized by a gradually spreading tremor, muscular weakness and rigidity, and a peculiar propulsive gait.

Etiology.—Advanced life, a neuropathic tendency, mental strain, heredity, and exposure to cold and wet are predisposing factors. It sometimes develops suddenly after intense mental or emotional excitement.

Pathology.—The pathology is unknown. The lesions found—degeneration of arterioles, perivascular sclerosis, pigmentation of ganglionic cells—are similar to those induced by senility.

Symptoms.—In some cases the onset is abrupt, but more commonly the disease develops insidiously. A tremor appears in the hand, usually in the forefinger and thumb ("pill rolling" movement), and gradually spreads until it involves all the extremities and occasionally the neck and head. At first the tremor may be paroxysmal, but as the disease advances it becomes almost continuous. Excitement increases it, but it is noteworthy that physical effort temporarily diminishes or checks it. The face becomes expressionless (mask-like), and the speech slow and measured. Later, muscular rigidity develops, and the head is bowed, the body bent forward, the arms flexed, the thumbs turned into the palms and grasped by the fingers, and the knees slightly bent. At this time the gait is characteristic. The steps are at first slow and shuffling, but they become progressively quicker and shorter until the patient is forced to run or come to a stop to prevent falling forward (*propulsion* or *festination*). Occasionally a tendency to fall backward (*retropulsion*) replaces festination. The rigidity and muscular weakness render all movements slow and stiff.

Intelligence is usually good. There is no anesthesia, but there are various manifestations of paresthesia, such as numbness and tingling and a sensation of heat. In some cases free perspiration is observed.

Diagnosis.—The tremor, rigidity, weakness, flexion of the body and members, lack of facial expression, and festination are the diagnostic features. In some cases the tremor is absent. Paralysis agitans must be distinguished from *disseminated sclerosis.* In the latter the tremor is absent when the patient is quiet, and is made worse by efforts to control it; cerebral symptoms are usually present; nystagmus is often noted; and the attitude and gait are entirely different from those of paralysis agitans.

Prognosis.—Recovery rarely, if ever, occurs. In some cases, after reaching a certain point, the disease remains stationary. The progress is slow and the duration indefinite.

Treatment.—Measures intended to improve the general nutrition are indicated; these include quiet living, adequate nourishment, abundant sleep, and the use of various tonics. Gentle exercise, active and passive, is desirable. Electricity is often harmful. Good results sometimes follow the use of hyoscin ($\frac{1}{200}$–$\frac{1}{150}$ grain, thrice daily) or of bromids. Dercum recommends parathyroid substance ($\frac{1}{20}$–$\frac{1}{10}$ grain, thrice daily).

OCCUPATION NEUROSES

An occupation neurosis is a form of muscular spasm induced by the frequent or prolonged execution of certain coördinated movements, and occurring only in the performance of work requiring those particular movements. The condition occurs chiefly in neurotic individuals as the result of overwork, and is observed most frequently in writers (*writer's cramp* or *palsy*), telegraph operators, pianists, typewriters, and seamstresses. It is probably due to exhaustion of the cerebral centers concerned in the execution of the movements which are affected by the spasm. In writer's cramp, which may be taken as a type of these disorders, the patient experiences a sense of fatigue or a dull ache in the wrist and hand and a tonic or clonic spasm of the muscles whenever he attempts to write. In some cases muscular weakness and tremors are also present. Neuritis can be excluded by the ability to execute all other movements involving the same muscles, and by the absence of tenderness along the course of the nerve and of muscular atrophy. The prognosis is good for recovery, if a sufficient period of rest can be enforced. Relapses, however, are common. Therapeutic measures comprise the absolute cessation of the activities that have led to the neurosis, massage, outdoor exercise, hydrotherapy, galvanism or the static breeze, and the administration of general tonics.

DISEASES OF THE MUSCLES AND JOINTS

MYALGIA

(Muscular Rheumatism)

Myalgia is an affection of the voluntary muscles characterized by pain, which is aggravated by movement. The pain varies in degree from a dull ache to an intense cramp. Malposition and even contractures may result from the efforts of the patient to avoid moving the affected muscles. The nature of the disease is obscure. It is probably in some cases a form of myositis or of neuritis affecting the nerve endings in the muscles. Various names have been given to it according to the different groups of muscles involved. The most frequent forms are: *Wry-neck* (torticollis) in which the head is fixed and rotated toward the unaffected side; *pleurodynia*, in which severe pain is experienced in the intercostal muscles upon coughing or turning the body; and *lumbago*, in which the lumbar muscles are painful and immobile. The condition may be acute or chronic.

Acute myalgia is usually due to overexertion or to chilling and wetting, especially when the body is overheated. Many writers lay stress upon a "rheumatic" or gouty diathesis. It is sometimes associated with acute articular rheumatism. Chronic myalgia is not infrequent in neurasthenia, anemia, gout, diabetes, chronic nephritis, and arteriosclerosis. Chronic lumbago may be the result of traumatism, pelvic disease, arthritis of the spine, sacro-iliac strain or relaxation, defective lateral or anteroposterior balance (spinal curvature), or flat-foot. In the diagnosis one must consider renal calculus, perinephritis, Pott's disease, aneurysm, and visceral cancer.

Treatment.—In mild cases it will suffice to put the affected muscles at rest. In pleurodynia this is accomplished best by strapping the affected side, and in lumbago by applying a large piece of adhesive plaster from the floating ribs to the iliac crests. In more severe cases it will be necessary to use dry or moist heat, liniments, or sinapisms and to administer a salicylate, perhaps with antipyrin or phenacetin.

℞. Salophen	ʒij
Acetphenetidini	ʒj
Codeinæ sulphatis	gr. ii.—M.

Faint chartulæ No. xii.
SIG.—One every three hours.

℞. Chlorali hydrati	
Pulveris camphoræ	āā ʒj
Methylis salicylatis	f℥iii.—M.

SIG.—To be rubbed over the painful parts.

A blister is occasionally required. If the pain is very severe an injection of morphin and atropin may be given. In lumbago, acupuncture sometimes yields excellent results. Hot packs and baths are often efficacious, but great care must be exercised to guard against exposure after their use. Persistent myalgia is sometimes favorably affected by massage and electricity.

In chronic cases treatment must be directed to the underlying cause. In lumbago of traumatic or arthritic origin a leather corset or steel back-brace may be required. In the sacro-iliac and static cases faulty habits of standing and sitting must be corrected and adequate support furnished by the use of flat-foot plates, high heels, properly fitted corsets or pelvic belts, etc. Recumbency for a part of the day is often desirable. Static or high frequency currents often afford temporary relief. After the more severe symptoms have subsided massage combined with proper passive and resistant exercises may be advantageously employed.

MUSCULAR DYSTROPHIES

Definition.—An atrophic condition of the muscles developing in early life and not dependent upon any lesion in the nervous system.

Etiology.—The disease usually manifests itself before puberty. It is more common in males than in females. It is frequently transmitted from generation to generation, and several members of the same family may be similarly affected.

Pathology.—No lesion in the cord or nerves is observed. Gowers regards the disease as of developmental origin. Microscopic examination of the muscles reveals atrophy of their fibers and an unnatural amount of fat and connective tissue. When the latter elements are considerably increased, a pseudohypertrophy results (pseudomuscular hypertrophy).

Varieties.—The following types are recognized: (1) Pseudomuscular hypertrophy; (2) Erb's juvenile dystrophy; (3) Landouzy-Dejerine type.

Pseudomuscular Hypertrophy.—This form begins in childhood between the second and seventh years. The first symptom to attract attention is weakness of the muscles; the child is awkward, stumbles, and in walking seeks support. As the paralysis increases the muscles, particularly those of the calf, thigh, buttock, and back, enlarge. The upper extremities are less frequently affected. When the child assumes the erect posture, the feet are wide apart, the belly protrudes, and the spinal column shows a marked curvature with the convexity forward. The manner of rising from the recumbent position is characteristic: The patient straightens himself either by grasping the knees or by resting the hands on the floor in front of him, extending the legs, and pushing the body backward. The gait is waddling in character.

The electric contractility of the muscles is gradually reduced, but the reaction of degeneration is not present. There are no fibrillary twitchings in the muscles. The knee-jerk

CONGENITAL AMYOTONIA

(Congenital Myotonia; Oppenheim's Disease)

This is a congenital disease characterized by flaccidity and loss of tone and power in the voluntary muscles, without absolute paralysis or atrophy of the muscles. The deep reflexes are diminished or lost and the electric excitability of the muscles is impaired. The condition usually disappears as the child grows older. It differs from the muscular dystrophies in being congenital and non-familial, and in the absence of wasting.

MYASTHENIA GRAVIS

(Asthenic Bulbar Palsy)

In this disease the muscles are rapidly exhausted upon slight exertion, but recover after a brief rest. All the muscles of the body may be affected, but those supplied by the cranial nerves are most frequently involved, the symptoms simulating those of true bulbar palsy (inferior polioencephalitis). There is no atrophy or reaction of degeneration, but with the faradic current, there is early exhaustion of the muscles (myasthenic reaction). The disease usually lasts for years, and remissions are common. Death may occur from disturbances of respiration or deglutition. The treatment consists in rest, careful feeding (nasal feeding if necessary) and the use of strychnin and other tonics. Massage and electricity are contraindicated.

ARTHRITIS DEFORMANS

(Rheumatoid Arthritis; Osteo-arthritis)

Definition.—A form of arthritis, chronic throughout or with an acute onset or acute exacerbations, leading to more or less permanent deformity and crippling of the joints, and probably dependent upon a local focus of infection. The disease is in no way related to either rheumatism or gout.

Etiology.—Arthritis deformans may occur at any age, but it is uncommon in the extremes of life. Females are attacked more frequently than males. An hereditary tendency to the disease is sometimes noted. Enfeeblement of the general health from mental strain, overwork, unsanitary surroundings, frequent pregnancies, etc., is an important etiologic factor. In many cases the disease can be definitely associated with some local infection, such as tonsillitis, otitis media, pyorrhea alveolaris, cystitis, gonorrhea, intrapelvic suppuration, or an infected wound. Occasionally trauma seems to stand in a causal relationship.

Pathology.—In one form (periarticular) the changes affect chiefly the soft tissues, and consist of serous effusion into the joint, thickening of the synovial membrane and surrounding structures, and slight erosion of the cartilages. In another form (atrophic) the cartilages show extensive destruction, the heads of the bones are bare and eburnated, and the shafts are rarefied. In a third form (hypertrophic) there is much new bone-formation (osteophytes) at the edges of the cartilages, and sometimes also in the surrounding fibrous tissues, causing great deformity. In many cases the changes are mixed and do not correspond exactly to any one form.

Secondary lesions are not uncommon. These include atrophy of the muscles, contractures, partial luxations, and localized neuritis.

Symptoms.—When the onset is acute the disease resembles rheumatism, but in contrast to the latter the smaller joints, especially those of the hands, neck, and jaw, are often involved, the inflammation rarely shifts from joint to joint, the temperature seldom exceeds 102° F., the pulse-rate is usually elevated out of proportion to the fever, endocarditis is exceptional, the salicylates are of little value, and, above all, there is a marked tendency to structural changes in the joints and muscular atrophy.

In the form with a chronic onset one or two joints are, as a rule, first attacked, and then the disease gradually becomes more or less general. Pain, swelling, and impaired mobility are, for a time, the chief symptoms, but sooner or later signs of structural changes show themselves. These comprise deformity, rigidity, and crepitation on movement. Muscular atrophy is also a constant feature, and very often contractures or partial luxations add to the deformity. In advanced cases the joints are usually fixed in a position of

sons, and usually affect
symptoms are persistent p
atrophy.

Spondylitis Deformans
term is applied to arthr
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sciatica and lumbago.

Heberden's Nodes.—The
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deformans.

Diagnosis.—The differe
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Prognosis.—Rheumatoic
gressive disease, sometimes
or becomes arrested. A co

Treatment.—In

oil are sometimes of service. Potassium iodid is occasionally beneficial. A number of English writers have spoken very favorably of guaiacol carbonate (10–20 grains thrice daily). Thyroid extract may be tried. Salicylates sometimes relieve pain, but have no influence on the disease.

Local Treatment.—Massage and graduated exercise, both passive and active, often accomplish much good. Superheated air-baths are sometimes very useful. Electricity is of little value. Rubefacient liniments have a palliative influence in mild cases. An ointment of mercury, belladonna, and ichthyol, or an ointment of iodin well rubbed into the affected part is sometimes efficacious. If the pain is severe and persistent, blisters, or light applications of the actual cautery may prove effective.

CHRONIC RHEUMATISM

Cases of rheumatic fever are not infrequently met with which drag on for weeks or months. To these the term *subacute rheumatism* is applicable. There is no proof, however, that rheumatism ever passes into a distinctly chronic condition or ever begins as a chronic affection. The many cases of polyarthritis with a tendency to chronicity and to permanent structural changes in or about the joints, which are usually referred to as chronic articular rheumatism, had better be regarded as examples of *rheumatoid arthritis*, and treated accordingly, until we possess a more accurate knowledge of their etiology and can classify them more definitely.

RAYN

(Sym

Definition.—A vasomo
anemia, congestion, and g

Etiology.—The cause
lieved to be dependent up
of central origin.

Symptoms.—In the firs
extremely pale, cold, and
a variable time the part b
painful (*local asphyxia*).
cold, and come and go
casionally the disease adv
congestion gives way to
as a finger on each hand
of the ears, are usuall
occur in, or replace, an at

Prognosis.—The attack
dangered.

Treatment.—Patients

ANGIONEUROTIC EDEMA

This affection, usually regarded as a vasomotor neurosis, characterized by the acute development of circumscribed wellings in the subcutaneous tissues and mucous membranes n various parts of the body, lasting from a few hours to several ays, and showing a marked tendency to recur. The face s the part most frequently affected. Coincident with the edema there may be disturbances of the gastro-intestinal tract such as nausea, vomiting, colicky pain, and diarrhea. In some instances the swelling is accompanied by burning and itching, and an outbreak of urticaria. Purpura may also occur.

The disease is most frequently observed in neuropathic subjects and is sometimes hereditary. The attacks may be excited by exposure to cold or the ingestion of certain articles of food (shell-fish, strawberries, etc.) poisonous to the individual. In a few instances death has resulted from edema of the larynx. Removal of the cause, if possible, regulation of the diet, and the judicious use of saline laxatives are important elements in treatment. Among special remedies that have been recommended may be mentioned calcium chlorid (10 to 15 grains thrice daily), nitroglycerin, strychnin, quinin, and arsenic.

HEAT STROKE

(Sunstroke; Thermic Fever; Insolation; Heat Exhaustion)

One of two conditions may ensue as a result of exposure to excessive heat—sunstroke or heat exhaustion. Lowered itality from other diseases, bodily fatigue, and alcoholic xcess are important predisposing factors. It is probable hat the excessive heat leads to the formation of certain toxic ubstances that disturb the heat-regulating centers or the asomotor centers in the brain.

Sunstroke (Thermic Fever).—This condition occurs after xposure to the sun's rays. Premonitory symptoms consist- g of headache, dizziness, nausea, and malaise are sometimes

persistent headache, impair
sensitiveness to high temper
mortality is probably not less
the pathologic changes con
ous organs, especially the b
blood, and parenchymatous
and kidneys. The conditio
occurred, with the intense fe
guish sunstroke from *apoplex*

Treatment.—The patient
ice-water and rubbed with ic
be employed. The intraveno
salt solution has proved ben
and others have found venes
effective in asphyxiated subj
not necessarily a contraindic
improves during the operation

Heat Exhaustion.—This c
to either natural or artificial
profound prostration, a subno
and collapse. Complete sync
semiconsciousness with mutt
is usually favorable. The

face, mental excitement, quickening of the pulse and respiration; then incoherent speech, delirium, dilated pupils, loss of coördination, subnormal temperature, vomiting, and, finally, stupor and coma. Not infrequently the coma is interrupted by convulsive seizures. In most cases, if the dose has not been too large, recovery follows in a day or two.

Chronic alcoholism is characterized by disturbed sleep, fine tremors, mental impairment, injection of the eyes, redness of the nose, and the symptoms of gastro-intestinal catarrh. When the habit is long continued, degenerative and cirrhotic changes in the heart, blood-vessels, liver, and kidneys are likely to develop.

A common complication of chronic alcoholism is *delirium tremens*, which is usually excited by temporary excess, an injury, or some acute intercurrent disease, especially pneumonia. It is manifested by great mental excitement, insomnia, incoherent speech, tremors, disordered intellect, and terrifying hallucinations of sight or hearing. The pulse is rapid and feeble, the appetite is lost, the bowels are constipated, and the temperature is slightly elevated. In favorable cases convalescence follows in a few days, but not rarely symptoms of the typhoid state develop or the patient sinks into a semicoma lasting several days or even weeks ("wet brain").

Among other sequels of dipsomania may be mentioned pneumonia, chronic meningitis, cirrhosis of the liver, arteriosclerosis, multiple neuritis, amblyopia, epilepsy, and dementia.

Diagnosis.—The *coma of alcoholism* must be distinguished from the coma of other diseases. The history, the absence of paralysis, the subnormal temperature, the fact that the patient can usually be aroused, the odor on the breath, and the absence of other cause will usually prevent an error in diagnosis.

Delirium tremens is recognized by the history, restlessness, delirium, tremors, and terrifying hallucinations.

Prognosis.—*In acute alcoholism* the prognosis is guardedly favorable. In *delirium tremens* recovery usually occurs unless the condition is complicated by trauma or pneumonia. In *chronic alcoholism* the outlook is not very favorable. Temporary improvement is only too often followed by relapse.

Treatment.—*Acute Alcoholism.*—The stomach should be emptied by the stomach-pump, a stimulating emetic, or the hypodermic injection of apomorphin (1/10 grain). If the coma persists and the pulse weakens, cardiac stimulants, like ammonia, strychnin, and digitalis, should be administered hypodermically. Douching and flagellation may also be employed to arouse the patient.

Treatment of Delirium Tremens.—As there has usually been a complete abstinence from food during the debauch leading to the delirium, easily digestible foods are always necessary, and the best are milk with lime-water and highly seasoned beef-tea. Sleep must be secured by chloral (20 grains), hyoscin (1/100 grain), potassium bromid (1 dram), or paraldehyd (1/2 to 1 fluidram). Active catharsis should be encouraged. If the pulse is weak, strychnin and digitalis will be found useful stimulants. In many cases physical restraint will be required; it may be secured by strapping the patient to the bed with sheets. Should profound stupor develop, the application of a blister to the back of the neck, a few light touches of the actual cautery, or lumbar puncture will often serve to arouse the patient.

Chronic Alcoholism.—It is necessary that alcohol shall be withdrawn; the rapidity with which this can be accomplished will depend on the circumstances. In most cases the temptation to drink is so strong that confinement in an inebriate asylum is essential to the success of the treatment. Various substitutes have been recommended for alcohol, among which may be mentioned bromid of potassium, chloral, hyoscin, and cannabis indica. These accomplish little beyond quieting the patient and securing sleep. The diet should be nutritious, and carefully adapted to the condition of the tomach, which is usually the seat of chronic catarrh. Tonics re often indicated. Graduated physical exercise is sometimes of decided value.

CHRONIC OPIUM POISONING

(Morphinomania)

Symptoms.—The symptoms resulting from the habitual use of opium are an irresistible craving for the drug, loss of flesh and strength, tremors, anemia, a peculiar sallow complexion, anorexia, deranged digestion, a tendency to diarrhea, disturbed sleep, mental depression, irritability, and a characteristic propensity for lying and deceiving.

Treatment.—Isolation in a special institution or asylum is almost imperative. As a rule, the drug should be withdrawn rapidly, but in aggravated cases not too abruptly, for fear of collapse. The diet should consist of nutritious, easily digested food. Strychnin, while it is without specific action, is often valuable for its stimulating effect. Bromids and cannabis indica are sometimes useful in ameliorating the distress that follows the withdrawal of opium. Sulphonal, paraldehyd, and chloretone are the best hypnotics. Massage, graduated exercise, and the Turkish bath are useful roborant measures in the convalescent stage.

CHRONIC LEAD-POISONING

(Plumbism; Saturnism)

Etiology.—Chronic lead-poisoning may be brought about y the too prolonged use of the salts of lead for medicinal urposes, but it is much more frequently induced in workmen ho are exposed to the fumes or dust of lead, or who handle he metal or paints containing it. It may follow, also, the ccidental introduction of lead into the system through rinking-water, articles of food, hair-dyes, and cosmetics. ccasionally it results from the use of water that has been

pation, a blue line on the g
teeth, due to the deposition o
and occasionally grave cerebra
tremors, and pains in the j
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The Paralysis.—This usuall
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and of the wrist—causing th
affected muscles ultimately a
of degeneration. Occasionall
such as the extensors of the
the adductors of the larynx.

Encephalopathies.—These ar
festations of plumbism, and
delirium, intense headache, a
the optic nerves.

Prognosis.—The outlook is
cases with encephalopathies.

Treatment.—Prophylaxis co
the use of respirators in lea
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and in the occasional use of E

The curative treatment co

PELLAGRA

Definition.—An endemic chronic disease characterized by igestive disturbances, erythematous roughness of the skin, specially on the exposed surfaces, and symptoms referable o widespread degenerative changes in the brain and spinal ord.

Etiology.—The disease prevails in southern Europe, es-ecially in Italy, and in the southern parts of the United tates. The cause is obscure; according to one view it is in ome way associated with the continuous use of spoiled Indian orn, according to another view it is an infection caused by a acterial or protozoal organism.

Pathology.—Degenerative changes are found in the cortical ells of the brain, the cells of the anterior horns of the cord, and in the nerve-fibers of the posterior and anterolateral columns. In long-standing cases there may also be some thickening of the meninges.

Symptoms.—The digestive disturbances are usually the first symptoms, and comprise stomatitis, abdominal pain, vomiting, and diarrhea. The cutaneous eruption appears in the spring and subsides in the winter. It is most commonly found on the dorsal aspect of the hands and feet, the chest and the neck, and takes the form of a desquamative erythema or dermatitis. The nervous features consist of headache, irritability, hallucinations and profound mental depression. In some cases the mental disturbance takes the form of true nelancholia, dementia or mania, and not rarely paresis of an taxic or spastic type develops.

Prognosis and Treatment.—Recovery is rare in well-eveloped cases. The course of the disease, however, is sually chronic, lasting from five to twenty years. Improve-ent of the environment and food supply of the people is he chief element in prophylaxis. Medication is mainly ymptomatic. Among special remedies, arsenic is the one ost commended.

Pallor as a permanent con
of anemia; but it should be
may be pale when the blood i
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Yellowness of the skin may
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pernicious anemia, and the *cac*

Whiteness of the Skin.—A
areas may be observed in *albi*

Dark-brown or gray disco
observed in the following
hemachromatosis (*bronzed dia*
silver nitrate (*argyria*), *melan*
malaria, *chronic jaundice*, *pern*
Graves' disease, *pellagra*, *and*
the last being a discoloration
cleanliness, and irritation caus

Blueness of the skin as a
an expression of cyanosis

elastic, and does not pit on pressure. In addition the features are peculiarly broadened and the mental power is impaired. *Edema*, or *dropsy of the subcutaneous tissues*, if extreme and persistent, may also cause induration.

GLOSSY SKIN

"*Glossy Skin.*"—This term was applied by Paget to indicate a smooth, atrophied, and shiny appearance of the skin. It is most frequently observed after inflammation or injury of the nerve-trunks. It is sometimes associated with an intense burning pain to which Mitchell has given the name *causalgia.*

ENLARGEMENT OF THE SUPERFICIAL VEINS

Enlargement of the superficial veins may result from chronic heart, lung, or liver disease or from the pressure of a tumor or aneurysm on deep-seated veins. As a general condition it may be congenital and result from occlusion of the deep-seated veins.

CUTANEOUS EMPHYSEMA

This term is applied to the presence of air or gas in the subcutaneous tissues. The condition is manifested by a diffuse swelling which yields a peculiar crackling sensation on palpation. It may result—(1) From the rupture through mechanical injury or ulceration of some air-containing structure, such as the trachea, lung, esophagus, stomach, or bowel; or (2), in rare instances, from the presence in the tissues of gas-producing bacteria, as in malignant edema.

ABNORMAL CONDITIONS OF THE NAILS

Atrophy of the Nails.—The nails may become dry, brittle, discolored, and cracked in organic disease of the spinal cord; after inflammation or injury of the peripheral nerves; after prolonged febrile diseases, such as typhoid fever, and in

result from injury; from
the spinal cord, as locor
affections involving the
eczema.

CUTANE

Macules.—Macules are
elevated nor depressed.

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conditions:

Syphilis.—Secondary sy
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and frequently cover the
by subjective symptoms;
the history or with the
scar of the chancre, bon
and sore throat.

Erythema multiforme m
eruption, but the macules
red papules or tubercles.
their preference for the ex
tribution; the short durati

apular rash that disappears in two or three days by slight squamation. The moderate fever, sore throat, swollen rvical glands, and history of contagion will assist in the iagnosis.

Toxic Rashes.—The ingestion of certain drugs (antipyrin, rsenic, quinin, therapeutic serums) and, in some persons, of ertain articles of food ("shell-fish," strawberries) is followed y the appearance of a red macular eruption.

Brown macules are observed in:

Lentigo, or Freckle.—The spots are small, and are found specially on exposed parts—face, neck, shoulders, and ands.

Chloasma.—Dark spots may result from irritation of the kin from the action of chemicals, heat, scratches, or blisters. hey are sometimes noted in general diseases, such as Addion's disease and syphilis. They also occur in primary affecions of the skin, as vitiligo, scleroderma, and leprosy.

Mole, or Nævus Pigmentosa.—Moles consist in congenital deposits of pigment on various parts of the body.

White macules are observed in:

Vitiligo.—Apart from the absence of pigment, the skin is normal in appearance and function. An excess of pigment s usually noted at the periphery of the white patches.

Leprosy.—In this condition there are structural changes in the skin and anesthesia in addition to the white appearance.

Morphea.—In the late stage of this affection the circumcribed patches are white or yellow. The structure of the kin is altered, and the periphery of the patches is distinctly yperemic.

Purpuric spots, petechiæ, or ecchymoses occur in many diseases and intoxications (see p. 176) and may also result from he bites of various insects (fleas, bedbugs, body-lice). In urpuric spots of parasitic origin the central bite-mark, the urrounding hyperemic zone and the presence of the insects re the diagnostic features.

character of the rash will indica

Rubella.—In some cases of r
diffuse. The history, slight fev
swelling of the postcervical gla

Local irritation from traum
poisonous plants, or drugs often

Erythema Intertrigo.—This oc
faces come in contact. The par
macerated. The condition exci

Eczema.—The skin is thicke
marked itching; the redness sh
is no fever.

Erysipelas.—The part is con
and swelling terminate in an a
ture is high.

Acne Rosacea.—This is a
appears on the face and is ass
dilated capillaries.

Pellagra.—The erythematous
and subside in the winter; they
feet, and neck; and they are
turbances, spasmodic tabes, an

Herpes.—The vesicles appear in groups or clusters; they are mounted on an inflammatory base; they show no tendency to rupture; they are frequently associated with burning or neuralgic pains; and they are distributed along the line of the nerve-trunks.

Dermatitis Venenata.—A vesicular eruption may result from contact with poisonous plants, such as the poison-ivy or poison-oak. The eruption usually appears on the exposed parts—face or hands; the part is red and swollen and there is intense itching.

Dermatitis Herpetiformis.—The vesicles are often irregular in shape; they appear in clusters; they are very tense; they show no tendency to rupture; they are frequently associated with other lesions—papules, pustules, and bullæ; they excite intense itching; and they appear in crops over a period of weeks or months.

Impetigo Contagiosa.—The eruption consists of small vesicles that subsequently enlarge until they reach the size of blebs; the vesicles appear in crops; are commonly discrete; are flat and umbilicated; are filled with a straw-colored fluid; they show no tendency to break, but dry up and form thin yellow crusts, and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from ten days to two weeks.

Vesicular Eczema.—The vesicles are quite small and are aggregated in patches; the intervening skin is red and thickened; the vesicles tend to break and pour forth a serous fluid that keeps the part moist; and the eruption is associated with intense itching.

Miliaria, or Heat-rash.—This may appear as an eruption of minute vesicles; they are always discrete; they are surrounded by red areolæ; they usually appear on the trunk; they are generally associated with pin-head papules; they show no tendency to rupture; and they excite a little burning and itching.

Scabies.—In this affection the vesicles are small; they are

elevation of the
size from a pea to an egg. Bl
ing conditions:

Impetigo Contagiosa.—The b
they contain a straw-colored
they are commonly discrete; th
but dry up and form thin ye
but little itching. The dise
inoculable, occurs especially ir
days to two weeks.

Dermatitis Herpetiformis.—T
ciated with papules, vesicles,
rounded by inflamed skin; t
show no tendency to break, b
brown crusts; and they excite

Pemphigus.—The bullæ app
itching; they lack an inflamn
they dry up, and leave behin
is generally chronic.

Syphilis.—The bullous syp
syphilis and very late in the a
of the bullæ soon become p
form dark-green, cone-shaped

contagion.

Papules.—A papule is the skin varying in size f are observed in the follow

Erythema Multiforme.— with macules and tubercle red or purple color; they a and they show no tendenc appear in the course of tw or no itching, but they are pains.

After the Use of Certain cubebs, and tar may prod tory will aid in the diagno

Eczema Papulosum.—Th aggregated, and often ass the skin is thickened; and

Miliaria, or Prickly He they are very often asso always remain discrete; th and they excite a little bu

Acne Vulgaris.—The pa

excite considerable itching. There is often a history of contagion.

Syphilis.—The papules are dark in color; they are widely distributed, being especially marked on the trunk and flexor surfaces of the extremities; they are usually associated with pustules; and they excite no itching. The history and the accompanying evidences of syphilis will aid materially in establishing the diagnosis.

Smallpox.—The papules are hard and have a shot-like feel; they soon terminate in umbilicated vesicles; they excite some itching, and they are associated with high fever, pain in the back, and often a history of contagion.

Measles.—The papules are small, and tend to run together to form crescentic-shaped patches; and they are associated with moderate fever, swollen cervical glands, coryza, conjunctivitis, and bronchitis. There is often a history of contagion.

Tubercles.—Tubercles are large, circumscribed, solid elevations of the skin varying in size from that of a large pea to that of a walnut. They are observed in the following conditions:

Erythema Nodosum.—The tubercles are large; they usually appear on the extremities; they are reddish-purple in color; they never suppurate; and they are associated with malaise, slight fever, and rheumatoid pains.

Erythema Multiforme.—The tubercles are usually associated with macules and papules; they are flat and are of a bright-red or purple color; they appear especially on the extremities, and they show no tendency to suppuration but gradually disappear in the course of two or three weeks. They excite no itching, but are often associated with prostration and rheumatoid pains. The disease is probably allied to erythema nodosum.

Lupus Vulgaris.—This may begin as a papule or nodule. It is especially observed on the face. The tubercles are of a pale-red color and are quite soft to the touch. [illegible]

are very firm and often
out ulcers that secrete a

Tinea Sycosis, or Bar
the hairy parts of the
Suppuration soon begins
the hairs become dry,
will reveal the trichophy

Leprosy.—One form of
The latter are of a pale-r
absorption or ulceration
anesthesia in the parts aff

Wheals, or Pomphi.—
of the skin, generally mo
in the center and pale-r
considerable itching. Th
conditions:

Urticaria.—The wheals
short duration; they may
and they excite intense it

Erythema multiforme,
ease), and certain insec
wheals.

Crusts.—Crusts consist
red, yellow, brown

the scalp. Itching is absent, and there are no evidences of inflammation.

Syphilis.—The crusts are thick; they are of a dark-brown or green color; and they are often associated with ulcers that freely discharge. The history and other evidences of syphilis will aid in the diagnosis.

Impetigo.—The crusts are thin and yellow, and they are associated with blebs that appear in crops.

Favus.—The crusts usually appear on the scalp: they are yellow, brittle, and cup-shaped: they are usually perforated by a hair, and have a peculiar musty odor.

Tinea Tonsurans, or Ringworm of the Scalp.—In neglected cases this affection may be associated with crusting. It is observed only in children. The grayish scales, the dry, brittle, and broken hairs projecting through the crusts, the alopecia, and the detection of the trichophyton are the diagnostic features.

Scales.—Scales are dry exfoliations from the upper layers of the skin. They are observed in the following diseases:

Squamous Eczema.—The scales are usually associated with papules; the underlying skin is red and thickened, and there is often marked itching.

Seborrhœa Sicca.—The scales are greasy, and the underlying skin shows no evidence of inflammation. The sebaceous follicles are often dilated.

Psoriasis.—The scales are dry, and are of a pearly-white color; they are associated with circumscribed, sharply defined, elevated, inflammatory patches. The extensor surfaces are especially involved. There is little or no itching.

Ichthyosis.—This affection begins in early life. The scales are dry, and are especially marked on the extensor surfaces. Itching is absent, and there is no evidence of inflammation.

Syphilis.—The scales are dry and are of a grayish color: they are usually associated with papules: and they are especially marked on the palms and soles. There is no itching.

The history and other evidences of syphilis will assist in the diagnosis.

Pityriasis Rosea.—The scales are found especially on the trunk, and are associated with small, rose-red macules. There is no itching. The disease runs an acute course of a few weeks' duration.

Ringworm.—The scales are dry and scant; they are associated with circumscribed red patches that tend to disappear in the center. There is often marked itching. Microscopic examination reveals the trichophyton.

Ulcers.—Ulcers are observed especially in the following diseases:

Syphilis.—The ulcers are deep; they have a punched-out appearance; they secrete an abundant offensive material; they often involve the bone; they extend rapidly; they are not painful, and the imperfect cicatrix which they produce is soft. The history and other evidences of syphilis will aid in the diagnosis.

Epithelioma.—This appears in late life; there is usually a single center of ulceration; the ulcer is irregular in shape; the edges are thickened and infiltrated; the secretion is scanty and bloody; the progress is somewhat slow, and there is often pain.

Lupus Vulgaris.—This usually appears in early life; there are often several centers of ulceration; the ulcers are usually superficial; the edges are not thickened; the progress is extremely slow; the bones are never involved; there is very little secretion, and soft papules often develop in the cicatrix, which is firm and contracted.

Simple ulcers may result from traumatism, the application of caustics, or the action of intense heat or cold. Ulcers are frequently observed on the legs of old persons in association with varicose veins. Simple ulcers may be recognized by the history, location, appearance, and absence of other causes.

Perforating Ulcer of the Foot.—This term is applied to a deep-seated ulcer appearing on the sole of the foot and most quently observed in locomotor ataxia. It usually begins a corn in the neighborhood of the great toe, and is generally sociated with anesthesia of the sole of the foot.

Decubitus.—This term is applied to the bed-sores that form ter the occurrence of grave cerebral or spinal lesions. They re usually observed on parts that are subjected to pressure, as the sacrum, buttocks, calves, and heels, and are preceded by erythema and vesication.

Definition.—Excessive sweati

Etiology.—As a general conc advanced tuberculosis and in o marked debility. Local hype observed in the hands, feet, an from some derangement of the Unilateral sweating of the face tumor pressing on the cervical s

Symptoms.—The primary sy and this often leads to intert Bromidrosis is often associated

Prognosis.—The disease is f ment.

Treatment.—The underlying should be treated. Internally, may be employed to diminish donna, picrotoxin, agaricin, or e

Local Treatment.—Dusting-p lycopodium with tannoform o lotions containing formaldehyd tannic acid, or alum are often u

℞. Pulveris acidi salicylici
Pulveris zinci carbonatis
Pulveris magnesii ustæ.

dried, and then carefully enveloped in strips of muslin that have been spread with diachylon ointment. The application should be made twice daily. In the dressing no water should be employed, but the feet must be carefully wiped and then dusted with starch or lycopodium before the ointment is reapplied. The treatment should be continued for from one to two weeks, after which the feet may be washed and the dusting-powder alone used.

BROMIDROSIS

(**Osmidrosis**)

This is a functional disturbance characterized by the secretion of sweat of an offensive odor. It is frequently associated with hyperidrosis, and the feet and axillæ are the parts commonly affected. The treatment is that of hyperidrosis. The *x*-ray is sometimes of service.

CHROMIDROSIS

This is a functional affection characterized by the secretion of colored sweat. The parts most frequently affected are the face and trunk; the most common colors are red and yellow. It is often associated with hyperidrosis.

SUDAMEN

Definition.—A cutaneous affection, characterized by the eruption of minute vesicles, resulting from the retention of sweat in the upper layers of the skin.

Etiology.—It is often observed in health in persons who perspire freely. It is frequently noted in febrile diseases that are associated with sweating, such as pneumonia and typhoid fever.

Symptoms.—Minute, irregular, translucent vesicles appear on the surface. They are not surrounded by inflammatory areolæ. They do not rupture, but dry up and are followed by slight desquamation. The affection has little significance, and treatment is rarely required.

S

Definition.—A functio
cessive secretion of seba
coating or scales. Tw
oleosa and seborrhea sicc

Etiology.—In many cas
the disease is associated w
By some it is regarded as

Seborrhea Oleosa.—T
on the face, particularly
bathed in an oleaginous
sebaceous follicles. From
The condition is frequent
comedo, and acne.

Seborrhea Sicca.—This
on the scalp, and constit
druff. Examination reve
thin, yellowish-gray, grea
the skin is pale, but from
come hyperemic or infla
the nutrition of the hai
results.

On the body, seborrhe

Diagnosis.—In *eczema* the skin is red and thickened; there is marked itching; and the scales are not greasy. In *psoriasis* the scales are dry and pearly and there are evidences of inflammation.

Prognosis.—This is favorable under prolonged and judicious treatment.

Treatment.—The general health may be impaired; hence tonics, such as iron, strychnin, and cod-liver oil, are often indicated. The gastro-intestinal tract will often require especial attention. Constipation should be relieved by diet, enemas, or mild laxatives.

Local Treatment.—Crusts should be removed by applications of oil, followed by shampooing with alcohol and green soap. When the scalp is thoroughly clean, one of the following remedies may be applied: sulphur, mercury, salicylic acid, carbolic acid, or resorcin.

℞. Ceræ albæ	ʒij
Petrolati liquidi	f℥ij
Aquæ rosæ	fʒvij
Sodii boratis	gr. x
Sulphuris	ʒij.—M.

Fiat unguentum.

Sig.—Apply at bedtime for several nights, then shampoo.

Or:

℞. Resorcinolis	ʒij
Olei ricini	♏xx
Spiritus myrciæ	
Alcoholis	āā f℥iij.—M.

Sig.—Fill an eye-dropper, introduce between the hairs, and subsequently rub in by means of a flannel rag.

Mild cases of facial seborrhea often yield to the following ointment:

℞. Hydrargyri ammoniati	gr. xx–xxx
Unguenti aquæ rosæ	℥j.—M.

Sig.—Apply at bedtime.

cleanliness are predisposin

Pathology.—The mate sebum, altered epithelium ably derived from withou material often reveals a but its presence is acciden

Symptoms.—The diseas of minute black or yellowis to the outlets of the seb slightly elevated, and wh filiform mass exudes, to been popularly applied. are the face, back, and associated with seborrhea

Prognosis.—This is us common.

Treatment.—Anemia, be treated by a careful and by the use of appr iron, quinin, cod-liver oil,

Local Treatment.—Larg means of a watch-key or a Softening and removal of the application of cloths wr

MILIUM

Definition.—An affection characterized by the appearance of small, pearly, non-inflammatory elevations, which result from the accumulation of inspissated sebum in ducts, the outlets of which have been occluded.

Symptoms.—It is usually observed about the face, and consists of a collection of small, round, pearly elevations, which vary in size from a pin-head to a millet seed. The contents of the distended duct cannot be squeezed out until an opening is made, and thus it differs from comedo. It is frequently associated with comedo and acne.

Treatment.—The lesions should be punctured, the contents squeezed out, and the interior touched with trichloracetic acid or tincture of iodin.

STEATOMA

(Wen)

Definition.—A steatoma, or wen, is a cyst resulting from the retention of secretion in a sebaceous gland.

Symptoms.—One or more rounded or oval elevations, varying in size from a pea to a large walnut, slowly appear on the scalp, face, or back. They are painless, rather soft, and when opened, are found to contain a yellowish-white, caseous mass.

Treatment.—The sac and its contents should be carefully dissected out. Simple excision and evacuation are always followed by a return of the cyst.

Simple active congestio
exposure to heat or cold,
x-rays, by traumatism, by
plants, soaps, etc., or by vari
It is characterized by diffus
and without thickening or
marked, there may be slig

The *treatment* consists in
sedative lotions or dusting

ERYTHEM

(

Definition.—Hyperemia i
surfaces of the skin.

Etiology.—It is common
and is especially noted whe
tion, as under pendulous ma
the thighs, and around the

Symptoms.—It is charact
and a feeling of soreness
epidermis may become ma
may ensue.

ERYTHEMA NODOSUM

(Dermatitis Contusiformis)

Definition.—An acute inflammatory disease, characterized by crops of large, bright-red nodes that, in the process of evolution, assume different colors, as in the fading of a bruise.

Etiology.—It is observed most frequently in children and young adults. Digestive disturbances are not rarely associated with it. It is closely allied to erythema multiforme, and an affinity appears to exist between both of these conditions and Henoch's purpura.

Symptoms.—There is a sudden eruption of bright-red nodes, varying in size from a pea to an egg. The extremities are most commonly affected. The advent is marked by malaise, headache, slight fever, and rheumatoid pains. At first the lesions resemble boils, but, unlike the latter, they do not suppurate, but gradually turn yellow, blue, and green, as a bruise.

Prognosis.—The outlook is favorable, recovery ensuing in from a few days to several weeks.

Treatment.—Saline laxatives and sodium salicylate are recommended. Locally, a lotion of lead-water and laudanum makes a soothing application.

ERYTHEMA MULTIFORME

Definition.—An inflammatory disease characterized by erythematous, papular, vesicular, or bullous lesions.

Etiology.—It is more common in women than in men. It usually develops in the spring or fall. Some form of intestinal intoxication appears to be the causative factor. Erythema nodosum, urticaria, angioneurotic edema, and Henoch's purpura are closely related affections.

Symptoms.—The disease is marked by an eruption, usually on the extremities, of macules, papules, vesicles, or, more rarely,

chronic character of c
prevent an error in diag

Urticaria.—In this dis
short time and are assoc

Prognosis.—The disea
a few days to several we

Treatment.—Rest anc
icylates are sometimes
relieved by saline laxa
carbolic acid followed by
effect.

(Hi

Definition.—An inflam
the eruption of whitish or
attended by severe itchin

Etiology.—Digestive d
posing factor. Idiosyncr
as fish, crabs, lobsters, cl
in some cases. Various
tar derivatives, and thera
association with certain

Pathology.—The disease apparently depends upon a disturbance of the vasomotor nervous system, spasm of the vessels first occurring, and then dilatation with outpouring of serum.

Symptoms.—The eruption appears suddenly and may be localized or more or less general. The lesions are firm, rounded, pinkish, or whitish elevations, surrounded by red areolæ (wheals). They last a few hours and are succeeded by new ones in other places. In some instances wheals of various shapes may be produced by drawing a blunt instrument or even the finger nail over the skin (dermographism).

Varieties.—*Urticaria Papulosa.*—In this form the wheal is followed by a lingering papule that is attended by considerable itching. It is most commonly observed in children.

Urticaria Hæmorrhagica.—The lesions are infiltrated with blood.

Urticaria Tuberosa (Giant Urticaria).—In this form the wheals may reach the size of an egg.

Diagnosis.—*Erythema Multiforme* and *Erythema Nodosum.*—In both of these affections the lesions last much longer and are free from itching.

Prognosis.—In acute cases recovery usually ensues in a few days. In chronic cases the outlook is uncertain unless the underlying cause can be ascertained and removed.

Treatment.—The cause should be sought for and removed if possible. In obstinate cases a diet composed for the most part of milk, farinaceous foods and vegetables, is, as a rule, most suitable. Laxatives, especially salines, are often serviceable. Intestinal antiseptics are sometimes efficacious. In the acute form a combination like the following may be prescribed:

℞. Acetphenetidini	gr. xl
Salophen	ʒi
Potassii bitartratis	ʒiss.—M.

Fiant chartulæ No. xii.
SIG.—One every four hours.

Among internal remedies worthy of trial in obscure cases may be mentioned: Calcium chlorid (5 to 15 grains thrice daily), quinin (5 grains thrice daily), pilocarpin (1/40 grain three or four times a day), and lacto-bacillary cultures. Externally lotions containing phenol and boric acid are very useful:

℞. Phenolis		fʒj
Glycerini		fʒss
Alcoholis		f℥j
Aquæ	q. s. ad	f℥viij.—M.

HERPES SIMPLEX

(Fever-Blisters)

Definition.—An acute, non-contagious disease, characterized by groups of small vesicles mounted on inflammatory bases.

Etiology.—Herpes is very common in febrile diseases, especially pneumonia, influenza, malaria, and cerebrospinal meningitis. Local irritation also predisposes to it. It is dependent upon a peripheral toxic neuritis.

Symptoms.—One or more clusters of small vesicles appear, usually on the face or genitalia. The vesicles are mounted on an inflammatory base, contain clear fluid, and show no tendency to rupture. Soon their contents become puriform, dry up, and form reddish-brown crusts that fall off in a few days. Burning and tingling precede and accompany the eruption.

Varieties.—If it appears on the face, it is termed *herpes facialis;* on the genitals, *herpes progenitalis.*

Diagnosis.—The history, the superficial character of the lesion, the burning pain, absence of glandular enlargement and the subsequent course will usually serve to differentiate herpes progenitalis from *venereal sores.*

Treatment.—In facial herpes an ointment of boric acid and menthol will be found efficacious:

℞. Acidi borici	gr. x
Mentholis	gr. iss
Petrolati	℥ ss.—M.

staphylococcus albus is usua

Pathology.—Acne lesio
by retained sebaceous mat
are commonly associated v

Symptoms.—There is
pustules, and comedones a
Pustules or papules predo
acute or chronic. New le
so that the disease usuall
jective phenomena are abs

Varieties.—*Acne Papulo*
the papular stage and adva

Acne Pustulosa.—In this
pustules.

Acne Indurata.—The in
base of the papule or pustul

Acne Atrophica.—In this
small scars or pits.

Acne Hypertrophica.—In
of connective tissue and the

Diagnosis.—The distribu
affection, the involvement
association with comedones
separate acne from all other

strychnin, and cod-liver oil are useful remedies. The special drugs which have been recommended are arsenic and calx sulphurata. Arsenic is best suited to the sluggish indurated forms; the calx sulphurata ($\frac{1}{10}$ to $\frac{1}{6}$ grain four times daily) to the pustular variety. Vaccine treatment, especially the conjoint use of the bacillus acne and the staphylococcus albus, is sometimes useful. ?

Local Treatment.—In the acute form mild applications should be employed, like the following calamin lotion:

℞. Pulveris zinci oxidi	ʒ iij
Pulveris calaminæ	ʒ ij
Glycerini	f ʒ j
Aquæ calcis	f ℥ vj.—M.

In chronic cases the sebaceous plugs should be removed by a watch-key and the pustules incised. Thorough washing with very hot water and green soap is also advisable. The best local remedies are sulphur, mercury, and resorcin.

℞. Calcis	℥ ss
Sulphuris sublimati	℥ j
Aquæ	f ℥ x.—M.

Boil together with constant stirring until the mixture measures six ounces and then filter.

SIG.—Apply at first well diluted and gradually increase the strength. (VLEMINCKX.)

Or:

℞. Sulphuris præcipitatis	ʒ j
Unguenti aquæ rosæ	
Petrolati liquidi	āā ʒ iv.—M.

SIG.—Apply night and morning. (VAN HARLINGEN.)

Or:

℞. Hydrargyri ammoniati	gr. xx–xl
Unguenti aquæ rosæ	℥ j.—M.

SIG.—Use night and morning.

Or:

℞. Hydrargyri chloridi corrosivi	gr. ss–ij
Tincturæ benzoini compositæ	f ʒ j
Emulsi amygdalæ amaræ	f ℥ iv.—M.

SIG.—Use locally.

hyperemia, dilatation of t
acne lesions.

Etiology.—Anemia, me
ances, exposure to extreme
are the usual predisposing

Symptoms.—The affect
vessels are dilated; the ski
lesions coexist. In adva
extremely large and lob
phenomena are usually ab

Diagnosis.—*Lupus Vulg*
pale-red papules, ulceratio
of the blood-vessels.

Prognosis.—Unless the
is curable under persistent

Treatment.—The genera
vulgaris.

Local Treatment.—Sulphu
remedies. Vleminckx's sol
Dilated vessels should be
hypertrophies may be remo

S

(Simple Sycosi

bearded region of the face, and is characterized by an aggregation of papules and pustules, each of which is pierced by a hair. When the lesions are discrete, the intervening skin is often quite healthy; but when they are close together, it is often infiltrated and hyperemic. During the papular stage the hairs are not loose, but firmly attached; during the pustular stage, however, they can be readily extracted. The pustules show no tendency to rupture, but dry to yellowish-brown crusts. Acute cases are associated with more or less burning and itching. If the disease persists, it may lead to destruction of the hair-follicles, and, as a consequence, to permanent alopecia.

Diagnosis.—*Eczema.*—The lesions in eczema excite severe itching, are not perforated by hairs, and are not confined to the hairy parts.

Tinea Sycosis, or Barber's Itch.—In this affection a red, scaly patch makes its appearance, and is followed by the development of large, deeply seated tubercles. The hairs soon become dry, brittle, and broken off, and can easily be extracted. In doubtful cases the microscope may be employed for the detection of the trichophyton.

Prognosis.—The disease is curable under prolonged and judicious treatment. Relapses are very prone to occur.

Treatment.—In acute cases soothing applications are indicated; thus the parts may be dabbed with black wash or a saturated solution of boric acid, and subsequently spread with oxid of zinc ointment. In chronic cases the crusts should be removed, and the hairs cut close or, preferably, shaved. It is advisable to puncture the pustules and to extract the hairs, so as to preserve the follicles. If the parts are not irritable, stimulating applications are useful, and one of the following may be selected:

℞. Sulphuris præcipitatis . . . . gr. xxx–ʒiss
Unguenti aquæ rosæ . . . . ℥j.—M.
SIG.—Apply twice daily.

Definition.—An acute
sebaceous gland or hair-f
puration.

Etiology.—Single boils
The appearance of boils i
indicative of impaired he
especially the staphylococc
is always essential to their

Diagnosis.—Furuncles r
buncles; the latter are singl
and have multiple openings

Treatment.—In furuncul
for and, if possible, remov
cod-liver oil, and hypophos
ous vaccines are sometime
(1/10–1/16 grain thrice daily
viceable. A solution of bo
may be applied locally. T
abort them:

℞. Ichthyol . . . . .
Unguenti hydrargyr
Extracti belladonnæ
SIG.—Apply locally an
plaster

Etiology.—The predisposing causes are the same as in furuncle. The condition is especially common in diabetes. The exciting cause is the invasion of several contiguous follicles by staphylococci.

Symptoms.—A dark-red, painful, flattened node appears, surrounded by a dusky-red area of induration. The entire lesion is often several inches in diameter. In a week or ten days suppuration begins, and the contents are discharged through several orifices. There is usually marked constitutional disturbance. The most common seats are the nape of the neck, back, and buttocks.

Prognosis.—Recovery usually ensues, but a fatal result is not uncommon in the aged and debilitated.

Treatment.—Generally tonics, such as quinin, iron, and whisky, are indicated. Opium may be required to relieve pain.

Local Treatment.—In the early stage they may sometimes be aborted by a central injection of 10 to 20 minims of a 5 or 10 per cent. solution of carbolic acid in glycerin. If not seen until abortion is too late, firm compression may be made by straps applied concentrically, leaving the central orifice free for the discharge of sloughs; an antiseptic dressing may be applied over the straps. As a rule, however, early surgical intervention is to be advised.

PSORIASIS

Definition.—A chronic inflammatory disease, characterized by red, scaly, sharply circumscribed, elevated patches.

Etiology.—Psoriasis usually develops in young adults. Heredity, the gouty diathesis, pregnancy, and lactation seem to predispose. It is as common in the robust as in the debilitated. It is non-contagious.

Pathology.—The lesions consist of a marked hyperplasia of the rete mucosum, thickening of the horny layer, and round-cell infiltration of the corium.

Symptoms.—Little red spots appear on the body and gradsurfaces are more affected

observed. Itching is sligh

able time the center of the

a spot of healthy skin that

of the lesion remains. Th

of months or years, impro

worse in the winter.

Diagnosis.—*Eczema.*—In

not sharply defined, but

rounding skin; there is m

decided discharge, and heali

of at the center as in psorias

Seborrhea.—In this affect

fined to the scalp and fac

scales are gray and greasy;

and lack the inflammatory

Papulosquamous Syphilod

symptoms of syphilis, the

scant scaling, and the speci

and soles will render the di

Prognosis.—The disease

relapse usually follows after

Treatment.—The general

In the gouty, alkalis are

line baths before local applications are made. The best local remedies are tar, oil of cade, chrysarobin, salicylic acid, resorcin, sulphur, and ammoniated mercury. Ointments of chrysarobin and mercury must not be used over too great an area.

℞. Olei cadini	fʒij
Adipis	℥j.—M.

SIG.—Apply night and morning.

Or:

℞. Chrysarobini	ʒj
Acidi salicylici	gr. x-xx
Ætheris	fʒj
Olei ricini	♏v
Collodii . . . q. s. ad	f℥j.—M.

SIG.—Apply with a camel's-hair brush and paint over with plain collodion. (STELWAGON.)

X-ray is sometimes useful in obstinate cases.

ECZEMA

(Tetter)

Definition.—An acute or chronic non-contagious inflammatory disease of the skin, characterized by multiform lesions—erythema, papules, vesicles, pustules, scales, and crusts—and associated with infiltration, itching, and more or less discharge.

Etiology.—It is most common in the young and in the aged. Digestive disturbances, debility, and gout predispose to its development. It may be due to external irritants such as cold, heat, the rhus-plant, hard soaps, certain dyes, etc.

Pathology.—The lesions consist of congestion, with a cellular and serous infiltration of the various layers of the skin.

Varieties.—Eczema erythematosum, papulosum, vesiculosum, pustulosum, squamosum, and rubrum.

Eczema Erythematosum.—This form consists in irregular patches marked by swelling, redness, and slight scaling, and accompanied by itching and burning. The most common seat is the face.

intense itching. The vesi
weeping surface that bec
crusts. In children it is m
on the extremities.

Eczema Pustulosum (**Ec**
sists in an aggregation of s
to the formation of thick
marked. It is frequently as
It is most commonly obser
nourished children.

Eczema Squamosum.—I
defined red patches accomp
differs from the erythemat
scaling. Its most common

When there is a marked
ping, this form is termed
a tendency to the format
termed *eczema verrucosum*.

Eczema Rubrum (**Eczema**
variety and is recognized b
more or less covered with
itching may be severe. In
the face, and in old people o

Diagnosis.—*Scabies.*—Th

Acne Rosacea.—The presence of acne papules and pustules and of dilated blood-vessels and the absence of itching will distinguish acne rosacea from erythematous eczema.

Seborrhea.—The greasy scales and the absence of itching and of all inflammatory symptoms will indicate seborrhea.

Sycosis.—The limitation of the lesions to the hair-follicles of the face and the absence of itching will distinguish sycosis from eczema.

Prognosis.—The outlook is favorable under persistent and judicious treatment.

Treatment.—*General Treatment.*—The health must be improved. Tonics are frequently indicated. In strumous children cod-liver oil may be of value. Disturbances of the gastro-intestinal tract are frequently present, and will require appropriate treatment. In the gouty the alkaline mineral waters, colchicum, and the salts of lithium are indicated. Constipation must always receive attention. Of the special internal remedies, arsenic is the most important; it is, however, indicated only in chronic cases in which bright redness, itching, and weeping are absent.

External Treatment.—In acute cases with marked inflammatory symptoms, soothing applications should be employed. A saturated solution of boric acid may be dabbed on for five or ten minutes, and may be followed by zinc ointment spread on lint; when there is much itching, carbolic acid is very useful:

℞. Acidi carbolici	ʒj
Glycerini	fʒij
Aquæ . . . . q. s. ad	f℥viij.—M.

SIG.—Apply locally.

The following is also frequently used:

℞. Zinci oxidi	℥ss
Pulveris calaminæ	℈iv
Glycerini	fʒiss
Liquoris calcis	f℥viij.—M.

SIG.—Shake and apply locally.

acid, tar, mercury, and resorcin

℞. Unguenti picis liquidæ
Unguenti zinci oxidi
Sig.—Apply twice daily.

Or:

℞. Hydrargyri ammoniati
Unguenti zinci oxidi

LICHEN RUBER, LICHEN SCROFU

Lichen Ruber.—This is a rare eruption of small, red, glazed, acu tendency to coalesce, and that a failure of general health. The and may prove fatal through ex

Lichen Planus.—This form is on the extremities of small, red, fl and, by coalescing, form dull- lesions have an angular outline, a first have a smooth and shiny app scaly. There is more or less itch general health. As the old lesi

Lichen Scrofulosis.—This is a chronic affection occurring chiefly in children of a strumous diathesis, and characterized by small pale-red or salmon-colored scaly papules. They tend to form in groups, and are most frequently observed on the trunk. Itching is absent. The disease runs a chronic course.

Treatment.—Remedies like iron, quinin, and cod-liver oil are indicated. Hebra recommends the last remedy as a local application.

PRURIGO

Definition.—A chronic inflammatory disease, characterized by a general eruption of minute, discrete papules, accompanied by marked itching.

Etiology.—It is most commonly observed in the poor and ill-fed of Europe, and is very rare in America. It develops in early childhood and persists through life.

Symptoms.—An eruption of small, discrete, deeply situated, pale-red papules appears on the body, especially on the back and extensor surfaces of the extremities. The skin is harsh, dry, and thickened, and covered with numerous scratch-marks induced by the intense itching. The disease usually persists through life.

Treatment.—The general health must be improved by good food and the use of nutrient tonics, such as iron and cod-liver oil. Frequent bathing, followed by ointments of tar, sulphur, or naphthol, gives relief.

DERMATITIS HERPETIFORMIS

(Herpes Gestationis; Duhring's Disease)

Definition.—A rare inflammatory disease, characterized by multiform lesions (vesicles, papules, pustules, etc.), forming in groups and accompanied by severe itching, and tending to run a chronic course.

Etiology.—Women are more commonly affected than men. Pregnancy, lactation, and menstrual disorders seem to exert a

frequently associated with erythe
marks.

Vesicular Form.—Groups of i
resembling herpes appear in crop
with erythema, pustules, and scrat

Pustular Form.—This resembles
are replaced by pustules.

Bullous Form.—Large, irregular
crops and tend to group. Vesicles
also frequently present.

Mixed Form.—Vesicles, erythe
papules, and blebs appear in associa
are attended with intense itching.

In the pustular, bullous, and
marked constitutional disturbance

Prognosis.—The disease is resist
continues indefinitely with remissi

Treatment.—Arsenic, quinin, a
the most useful remedies. Laxati
Locally, lotions of boric acid or
dusting-powder of zinc oxid and
to allay itching. Liquor carboni
ounce of camphor water, gradua
the ounce) is also useful.

Treatment.—The removal of the cause and the application of soothing remedies will usually suffice.

Dermatitis Venenata.—This term is applied to inflammation of the skin resulting from the application of vegetable, animal, or chemical irritants. Notable examples of this form of dermatitis are observed in susceptible persons after exposure to the influence of poison-ivy (*Rhus toxicodendron*), poison-oak (*Rhus venenata*), or poison-sumach (*Rhus diversiloba*).

Symptoms of Rhus-poisoning.—The affection resembles acute eczema, and may appear in a few hours or not until the lapse of several days after exposure to the plant. It is usually observed on the face or hands. The part becomes red and swollen, and soon minute papules and vesicles appear. The eruption gives rise to considerable burning and itching. As a rule, it subsides in a few days, but in patients with sensitive skin it may linger for several weeks.

Treatment.—The part should first be bathed with Castile soap and tepid water, and then treated with some sedative lotion or ointment. Black wash may be dabbed on, and zinc ointment subsequently applied; or a saturated solution of boric acid may be followed by zinc ointment. If there is marked itching, a weak solution of carbolic acid (1 dram to 1 pint) is useful. The fluid extract of grindelia robusta has been highly recommended; it may be applied in the strength of an ounce to a pint of water.

Dermatitis Calorica.—This term is applied to the inflammation of the skin resulting from extreme heat or cold. *Pernio*, or *chilblain*, is characterized by redness, swelling, intense burning, and itching, and results from a sudden change from a low temperature to a high temperature. *Frost-bite* is characterized by congelation; the part is of a dull-white color and is anesthetic; subsequently inflammation or gangrene develops.

Burns and *scalds* result from the application of dry and moist heat respectively, and are divided into degrees according to the depth to which the destructive process extends.

Treatment.—In *pernio*, or *chilblain*, the part should first be

℞. Acidi carbolici .
Petrolati . . . .
Sig.—Spread on lint a

Dermatitis Medicament
various cutaneous eruptio
certain drugs.

Belladonna or Atropin.
erythematous rash resembl
the punctiform character o
the face, neck, and chest,
the throat, rapid pulse, and
pupils.

Cubebs.—This drug some
ciated with minute papules.

Copaiba.—The rash may
urticaria.

Potassium Bromid.—The
sists of papules and pustule

Potassium Iodid.—The
papular, pustular, urticarial
eruption resembles acne, bu
and widely distributed over

Arsenic.—The eruption

[illegible]
often indicated.

Local Treatment.—The

stimulating ointment appli

℞. Hydrargyri ammo
Unguenti zinci oxi

PE

Definition.—A rare, acut
characterized by the erupt
blebs.

Etiology.—General debili
is a predisposing factor.
America.

Varieties.—Pemphigus a
phigus foliaceus.

Pemphigus Acutus.—Fe
mark the onset. Blebs, va
and containing clear or t
crops and usually develop f
recovery ensues in the cours

Pemphigus Vulgaris.—T
course, and is characteriz

subsequently detached, leaving behind a slightly pigmented spot. No part of the body is exempt; and as one set of blebs disappears, new ones rapidly develop, and so the disease continues for many years.

Pemphigus Foliaceus.—This rare and grave form of pemphigus is characterized by crops of blebs, which are flaccid and filled with a turbid fluid almost from the beginning. They soon rupture and form thick crusts, which, separating, leave behind red weeping surfaces. The crops follow each other in rapid succession, and at times the whole body may be covered with blebs and scabs. The disease may last several years, death ultimately resulting from exhaustion.

Diagnosis.—*Bullous Syphiloderm.*—The history, the associated symptoms of syphilis, the thick, yellow, stratified crusts, and the underlying ulceration will serve to separate this affection from pemphigus.

Impetigo Contagiosa.—The acute course, the contagious and auto-inoculable character of the affection, and the umbilication of the blebs will separate impetigo contagiosa from pemphigus.

Prognosis.—The duration and outcome are uncertain. Cases with purulent or hemorrhagic lesions are extremely grave and pemphigus foliaceus is almost always fatal.

Treatment.—The diet should be nutritious, but carefully adapted to the condition of stomach. The patient should be placed under the best hygienic conditions. Tonics, such as iron, quinin, phosphorus, cod-liver oil, and strychnin, are usually indicated. In many cases arsenic is a valuable remedy.

Local Treatment.—The blebs may be punctured and subsequently dressed with an ointment of boric acid.

IMPETIGO CONTAGIOSA

Definition.—An acute, contagious, inflammatory disease, characterized by flat, yellowish blebs that dry up and form thin, yellow, lamellated crusts.

into flat, slightly umbilicated
a pea to a large walnut. Th
Itching is slight or entirely a
moderate fever with its associa
the blebs dry up and form thi
separating, leave a slightly exc
contagious, and the lesions are

Diagnosis.—*Eczema.*—In t
deeper, more confluent, excite
ciated with inflammation and
skin.

Prognosis.—The disease ter
days to two or three weeks.

Treatment.—A slightly stin
following is useful:

℞. Hydrargyri ammoniati .
Adipis . . . .
Sig.—Apply to the surface

MILL

(Prickly

and itching. It is usually associated with free perspiration. Recovery occurs within a few days or weeks.

Diagnosis.—*Eczema papulosum* differs from miliaria in that the papules are larger, appear more gradually, disappear more slowly, and excite intense itching.

Eczema vesiculosum differs from miliaria in that the vesicles are large, disappear more slowly, show a tendency to break, and are associated with marked itching.

Sudamen differs from miliaria in that it lacks all inflammatory characteristics.

Treatment.—The general health may require attention. The diet should be light and easily assimilable. Constipation should be relieved by saline laxatives. Locally, a lotion of boric acid, followed by a simple dusting-powder, is usually all that is required.

℞. Pulveris amyli	ʒvj
Zinci oxidi	ʒiss
Pulveris camphoræ	ʒss.—M.

SIG.—Dusting-powder. (HARDAWAY.)

POMPHOLYX

(Dysidrosis)

Pompholyx is a very rare disease, usually observed in those who perspire freely, and characterized by an eruption of deeply seated vesicles that resemble sago-grains imbedded in the skin. The vesicles most commonly appear on the hand, especially between the fingers, and gradually increase in size until they reach the dimensions of blebs. They show no tendency to rupture, but dry up, and are followed by extensive desquamation of the cuticle. The eruption often excites considerable pain and tenderness. The disease usually disappears in the course of a few weeks, but is prone to recur.

Treatment.—General tonics, such as iron, strychnin, and arsenic, are often indicated. Locally, sedative lotions or ointments should be employed.

ALBINISM

Definition.—A congenital deficiency of pigment.

Etiology.—Beyond heredity, no cause is known. Partial albinism is more common in the negro.

Symptoms.—In complete albinism the skin is white; the hair is thin, soft and very light in color; the pupils appear red, the eyes are very sensitive to light, and the iris and choroid are deficient in pigment.

VITILIGO

(Leukoderma)

Definition.—An acquired cutaneous affection, characterized by milk-white patches that are surrounded by areas of increased pigmentation.

Etiology.—The disease seems to be more common in the tropics and in the colored race. The condition probably results from disturbed innervation.

Symptoms.—Milk-white spots appear on the body and grow very slowly; their borders usually reveal an increase of the normal pigment. Apart from the absence of pigment the skin is normal. The outlook for cure is unfavorable.

Diagnosis.—*Morphea.*—The initial hyperemia and the subsequent atrophy of the skin will serve to distinguish morphea from vitiligo.

Anesthetic Leprosy.—The subjective symptoms, the atrophy of the tissues, and the anesthesia will separate leprosy from vitiligo.

Treatment.—Tonics and local stimulants may be tried. Among the latter, electricity, blisters, and irritating ointments have been recommended.

ALOPECIA

(Baldness)

Etiology.—(1) Baldness may be congenital; in these cases it is usually partial. (2) It may be an expression of senility, in which case it usually begins on the crown or brow, and is associated with more or less atrophy of the scalp. (3) It may occur early in life, as an idiopathic affection arising without obvious cause. (4) It may result from general diseases, like syphilis and the fevers. (5) In early life it is often due to some local disease, especially seborrhea.

Prognosis.—In congenital, senile, and idiopathic alopecia the prognosis is unfavorable. In the alopecia of general diseases the prognosis is usually favorable. In alopecia resulting from seborrhea much can be accomplished by persistent and judicious treatment.

Treatment.—The general health should be improved. Shampooing every one to three weeks with warm water and Castile soap is to be recommended. One of the following local stimulants may be prescribed: Cantharides, quinin, alcohol, capsicum, sulphur, or carbolic acid.

℞.	Tincturæ cantharidis	f℥j
	Acidi carbolici	ʒj
	Olei ricini	ʒiss
	Spiritus myrciæ	
	Spiritus lavandulæ . . . āā	f℥ij.—M.

Or:

℞.	Tincturæ cantharidis	fʒij
	Quininæ sulphatis	gr. x
	Glycerini	fʒij
	Olei rosmarini	gtt. v
	Spiritus myrciæ . . . q. s. ad	f℥v.—M.

If there is much dandruff, the following lotion will be found useful:

℞.	Resorcinolis	ʒij
	Acidi salicylici	gr. xxx–ʒj
	Olei ricini	♏x–xx
	Alcoholis . . . q. s. ad	f℥vj.—M.

(SCHAMBERG.)

ALOPECIA AREATA

(Alopecia Circumscripta)

Definition.—Baldness appearing in circumscribed patches without any obvious lesion of the skin.

Etiology.—The cause is unknown. Some regard it as of

the scalp is the most frequent se
other hairy parts, as the eyebrows

Diagnosis.—*Ringworm.*—Ringw
is characterized by elevated sca
project dry, brittle, broken hairs
doubt in the diagnosis, the micro
detect the trichophyton.

Prognosis.—In the majority of
prolonged and persistent treatme
the less favorable the prognosis.

Treatment.—General tonics, suc
strychnin, are usually indicated.
be stimulating and consist in
electricity, friction, rubefacient
containing chrysarobin, tar, sulph

℞. Tincturæ cantharidis
Tincturæ capsici . . . .
Olei ricini . . . .
Spiritus rosmarini . . .
Alcoholis . . . .

Or:

℞. Betanaphtholis . . . .
Petrolati . . . .

LENTI

Symptoms.—Exposed parts—the face, shoulders, arms, and hands—are mostly affected. The patches vary in color from yellow to dark brown and range in size from a pin-head to a pea.

Prognosis.—Freckles can be removed, but they always return.

Treatment.—One of the best remedies is the bichlorid of mercury in solution or ointment.

℞. Hydrarg. chlor. corros	gr. ii-iv
Alcoholis et aquæ. . . . . āā. q. s. ad	f℥iv.—M.

Sig.—Apply twice daily.

CHLOASMA

Definition.—An abnormal deposition of pigment in the form of large brown or liver-colored patches.

Etiology.—It may result from the application of external irritants; from general diseases like .tuberculosis, malaria, exophthalmic goitre, and Addison's disease; or from pregnancy or uterine affections.

Symptoms.—The affection consists in the appearance—especially on the face—of large, round, or irregularly shaped brownish or blackish patches. Apart from the discoloration the skin is normal.

Prognosis and Treatment.—If the cause can be removed. the prognosis is favorable. The best local remedies are bichlorid of mercury, ammoniated mercury and hydrogen dioxid.

℞. Hydrargyri chloridi corrosivi . .	gr. iij-xij
Acidi acetici diluti	f℥ij
Sodii boratis	gr. xl
Aquæ rosæ . . . . q s. ad	f℥iv.—M.

Sig.—Apply night and morning.

HARDY.

KERATOSIS PILARIS

(Lichen Pilaris)

Definition.—Small, papular elevations resulting from hypertrophy of the epidermis surrounding the outlets of the hair-follicles.

Diagnosis.—In *cutis anser*
are transient and have the co

Treatment.—In most cases
frequent bathing with soap,
In obstinate cases an ointme
the ounce) may be applied af

MOLLUSCUM

(**Molluscum Contagiosu**

Definition.—A cutaneous
appearance of discrete, wax-
from a pin-head to a pea, and
rose-pink.

Etiology.—The disease is us
frequently affects several me
school, or asylum. It is prob

Symptoms.—Small white o
appear, especially on the face.
rarely abundant. The cente
and reveals a dark spot that
the follicle. At first the lesi
grow old they become soft. W
a soft cheesy material. Aft

are often indicated. The lesions should be incised, the contents expressed, and their bases touched with trichloracetic acid or nitrate of silver. Ointments of mercury and sulphur have also been recommended.

CALLOSITAS

(Callus; Keratoma; Tylosis)

Definition.—A thickened, horny condition of the skin resulting from hypertrophy of the corneous layer of the epidermis.

Etiology.—Constant irritation from friction or pressure is the chief cause; hence it is frequently seen on the feet from the rubbing of shoes, and on the hands from the friction of tools.

Symptoms.—The condition is characterized by the appearance of hard, thickened, grayish masses, which gradually merge into healthy skin. The soles and palms are the parts most frequently affected. When slight, it causes little inconvenience, but occasionally it becomes fissured and painful.

Prognosis.—It yields rapidly to treatment when the cause is removed.

Treatment.—If excessive, the parts should be soaked and the thickened epidermis pared off. One of the best remedies for softening the horny overgrowth is salicylic acid; it may be applied in the form of an ointment or plaster, or in collodion.

℞. Acidi salicylici	ʒi
Olei ricini	♏x
Collodii	f℥j.—M.

Sig.—Apply night and morning.

CLAVUS

(Corn)

Definition.—Clavus is a circumscribed thickening of the epidermis usually appearing on the feet.

Etiology.—Corns generally result from the friction of ill-fitting shoes.

Symptoms.—Small, circumscribed, horny elevations appear upon the feet and often excite severe pain. When bathed in perspiration, they become more or less macerated, and in this condition constitute the so-called *soft corn.*

Treatment.—A radical cure requires the use of well-fitting shoes. The corns may be removed by soaking, paring, and the application of some mild caustic like salicylic acid.

℞. Acidi salicylici	gr. xxx
Collodii	f℥ss.—M.

Sig.—Apply night and morning for several days, and then soak in hot water.

℞. Acid salicylici	ʒj
Emplastri plumbi	
Petrolati	āā ℥ss.—M.

Sig.—Apply night and morning.

For soft corns a dusting-powder of salicylic acid (10 grains), boric acid (1 dram) and powdered talcum (½ ounce) may be used.

CORNU CUTANEUM

(Cutaneous Corn)

Definition.—A circumscribed, projecting outgrowth resulting from hypertrophy of the epidermis.

Symptoms.—Horns usually appear on the face, scalp, or penis, and are usually observed in the old. They consist of dry, rough, horny, more or less conic projections, which vary in length from a few lines to several inches.

Treatment.—The horn should be excised and the base subsequently cauterized.

VERRUCA

(Wart)

Definition.—A wart is a circumscribed elevation resulting from hypertrophy of the papillæ and epidermis.

Etiology.—The cause is obscure. A bacterial origin has been suggested. They are most frequently observed in children.

Symptoms.—*Verruca vulgaris*, or common wart, is generally observed on the hands of children. It consists of a firm, circumscribed elevation, varying in size from a millet-seed to a pea.

Verruca plana, or flat wart, is a circumscribed, flat, pigmented elevation usually observed on the backs of old persons.

Verruca Filiformis.—This is a thread-like overgrowth, and is generally observed on the soft parts, like the face and neck.

Verruca Digitata.—This form is made up of numerous branches, and is generally observed on the scalp.

Verruca Acuminata, or Venereal Wart.—This appears in groups about the genitalia. It is soft, red in color, and highly vascular. It may be dry or moist, according to its location; the latter condition often gives rise to a peculiarly offensive odor.

Treatment.—Ordinary warts may be removed by excision, caustics, or electrolysis.

Venereal warts should be bathed in some antiseptic solution and subsequently dusted with calomel, iodoform, or boric acid.

NAEVUS PIGMENTOSUS

(Mole)

Definition.—A circumscribed deposit of pigment, usually associated with hypertrophy of cutaneous structures.

Etiology.—Moles are usually congenital.

Symptoms.—The neck, face, and trunk are favorite localities. The nevi vary in number from one to several hundred; in size, from a millet-seed to a filbert; and in color, from yellow to black. When the surface is smooth, the growth is termed *nævus spilus;* when the surface is covered with hair, it is termed *nævus pilosus;* when the surface is warty, it is termed *nævus verrucosus;* and when there is much overgrowth of connective tissue, it is termed *nævus lipomatodes.*

Treatment.—They may be removed by excision, the application of caustics, or by electrolysis.

Symptoms. The skin is
covered with adherent polyg
more or less hypertrophied.
extremities are the parts most

Diagnosis.—The absence of
separate ichthyosis from *squa*

Prognosis.—The disease is
be rendered comfortable by a

Treatment.—The scales ma
or by applications of green so
pliable by rubbing in an oint
to the ounce) or sulphur (20
extract has been recommende

HYPERT

(Hi

Hypertrichosis, or hypertr
or general. The term is ap
overgrowth of hair, but to a gr
as on the faces of young wom

Treatment.—The hair ma

Pathology.—Examination of th
hypertrophy of the connective tis
tion and dilatation of the lymphati

Symptoms.—It usually begins
erysipelatoid inflammation. The
painful; the lymphatics may be tr
beneath the skin; and with these
more or less fever. After each att
enlarged, until finally it presents
appearance: it is enormously swol
roughened, and pigmented; and
prominent. The regions generally
genitals. In elephantiasis of the
the hypertrophied mass may wei
100 pounds.

Prognosis.—In the early stage t
but when fully established, it is in

Treatment.—The acute inflam
treated by rest and the application
lead-water and laudanum. Subs
tions may be employed, and the
the view of promoting absorpti

Etiology.—It usually results from local injury, though it is claimed that it may arise spontaneously. Certain families and individuals are especially predisposed. It is more frequent in the colored race.

Symptoms.—It begins as a pale-red nodule, which slowly increases in size and sends out claw-like processes. From its resemblance to a crab it has been termed keloid. It is firm, elastic, slightly elevated, sharply defined, and ranges in size from a small bean to a growth as large as the hand. It sometimes excites pain and itching, but usually subjective phenomena are absent. The regions most frequently involved are the chest and back.

Diagnosis.—Keloid may be distinguished from a *hypertrophied scar* by the fact that the latter does not extend beyond the limits of the injury.

Prognosis.—The growth is persistent and tends to return after removal.

Treatment.—It may be removed temporarily by excision, electrolysis, or caustic pastes.

FIBROMA

(Fibroma Molluscum, von Recklinghausen's Disease)

Fibroma is a connective-tissue new-growth characterized by the development of variously sized soft or firm tumors in the skin and underlying tissues. The tumors are painless, range in size from a pea to a hen's egg, and are frequently numerous. The overlying skin may be normal in appearance or slightly hyperemic.

In von Recklinghausen's disease the fibromas are associated with painful neurofibromas, springing from the nerve-trunks; areas of brownish pigmentation; mental deterioration, and in some cases difficulty in speaking. Both conditions are intractable to all treatment except operative measures.

elevated, dark-red tumor that ra
one as large as the hand. It is o

Angioma Simplex (**Capillary**
—This form is also congenital, an
of capillaries. It is non-elevated
color, and may cover an area of s
found on the face, and constitutes
mother's mark.

Telangiectasis.—This form is a
dilated or newly developed capilla
red dot from which branch dilated
associated with acne rosacea; it
a gouty diathesis and in those m

Treatment.—Cavernous angio
ligation, excision, or electrolysi
telangiectasis are best treated by

XANTHO

(Vitiligoidea; Xa

Definition.—A circumscribed c
appearing as flat patches or tuber

xanthoma tuberosum, which may appear on the neck, shoulders, trunk, or extremities, and consists of small, elastic, and yellowish-colored nodules.

Treatment.—These growths may be removed by excision, electrolysis, or caustics.

LUPUS ERYTHEMATOSUS

(Seborrhœa Congestiva)

Definition.—Lupus erythematosus is a new-growth resulting from a cellular infiltration of the skin, and characterized by circumscribed, red patches that are more or less covered with yellowish-gray adherent scales.

Etiology.—It is a disease of adult life and is more common in women than in men. It frequently arises from disorders of the sebaceous glands, as seborrhea or acne.

Pathology.—By many it is regarded as a chronic dermatitis which originates in the sebaceous glands.

Symptoms.—The disease usually manifests itself on the face, in the region of the nose, and appears as small, red, slightly elevated papules, which are more or less scaly. An erythematous patch is gradually formed by the coalescence of these papules. The periphery of the patch is elevated and sharply defined, while the center is depressed and atrophied. The ducts of the sebaceous glands are dilated and often filled with sebum. The disease spreads very slowly, shows no tendency to ulceration, and rarely excites any subjective symptoms.

Diagnosis.—The location, the sharply defined red patch with an elevated margin and depressed center, the slight scaliness, the dilated sebaceous ducts, the chronic course, and the absence of ulceration are the diagnostic features.

Lupus Vulgaris.—This affection begins earlier in life, is characterized by tubercles and ulceration, and lacks involvement of the sebaceous glands.

Prognosis.—The disease is rebellious to treatment and a complete cure somewhat exceptional.

few days and then applying the fol

℞. Zinci sulphatis
Potassii sulphidi . . . . .
Aquæ . . . .
Alcoholis . . . .
Sig.—Shake well, dab the parts
and allow to dry on.

In sluggish cases ichthyol (20 pe
40 per cent. in water as a lotion) is

Treatment by the *x*-ray or carb
followed by excellent results in som

LUPUS VULG

(Lupus Exede

Definition.—A local manifestati
acterized by soft red tubercles t
ulceration and scarring.

Etiology.—The disease usually be
adult life. It is comparatively rare
common in Austria and Germany.
tubercle bacillus.

Symptoms.—Lupus vulgaris m
itself on the face especially near

the ulcer is spreading, other parts are being filled with shriveled cicatricial tissue which in turn is often the seat of new tuberculous nodules.

Diagnosis.—*Epithelioma.*—Epithelioma is a disease of advanced life; it begins as a firm, wax-like nodule; the resulting ulcer starts from a single point; its borders are distinctly elevated and hard; it secretes a blood-streaked fluid: and it is often painful.

Syphilis.—The age, history, associated evidences of syphilis, the rapid course, the deep ulcers, the abundant offensive discharge, and later, the involvement of the bones. are the diagnostic features.

Prognosis.—The disease is rebellious to treatment, and relapses are common.

Treatment.—General tonics, such as iron, arsenic. phosphorus, and cod-liver oil, are usually indicated.

Local Treatment.—The growth may be removed by cauterization, cureting, excision, or electrolysis. One of the following caustic applications may be employed:

℞. Acidi salicylici	ʒss–j
Collodii	f℥j.—M.

Or:

℞. Acidi pyrogallici	ʒij-iij
Vaselini et cerati resinæ . . āā q. s. ad	℥j.—M.

(STELWAGON.)

Often the best results are obtained by cureting and subsequently applying caustics.

Both Finsen light and *x*-ray therapy have also been used with considerable success in the treatment of lupus.

SYPHILIS CUTANEA

The *secondary symptoms* appear between the first and the fourth month following the chancre, and are characterized by a symmetric arrangement, a coppery color, polymorphism (many forms *at the same time*), and an absence of itching.

They are usually associated with certain general symptoms, such as sore throat, pain in the bones, loss of hair, enlargement of the lymphatic glands, and failure of health.

The *tertiary symptoms* appear in from six months to several years after the primary sore. They are, as a rule, localized, are tubercular, gummatous, or ulcerative in form, and tend to group.

Macular Syphiloderm.—This is a secondary manifestation, and consists in a general eruption of dark-red macules, varying in size from a millet-seed to a ten-cent piece.

Diagnosis.—*Measles.*—The absence of fever, of catarrh, of a crescentic arrangement, together with the history, will prevent an error in diagnosis.

Papular Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of large or small, dull-red papules. A few pustules are also frequently present. It pursues a chronic course, finally disappearing by desquamation, and leaving behind slight pigmentation.

Diagnosis.—The history, distribution, dark color, and the presence of pustules will separate it from keratosis pilaris, papular eczema, and lichen ruber.

Tuberculous Syphiloderm.—A late manifestation, characterized by a localized eruption of dark-red, shiny papules varying in size from a pea to a large bean. By some these tubercles are regarded as gummatous in character. They pursue a chronic course and finally disappear by absorption or ulceration. The ulcers thus formed, when single, are round, punched out, and frequently covered with crusts; when they coalesce, they form a serpiginous sore that pours forth a thick yellowish discharge.

Diagnosis.—*Lupus Vulgaris.*—This occurs in earlier life; it pursues an extremely chronic course; the ulcer is superficial; the tubercles are soft, and frequently redevelop in the scar tissue; the secretion is scant; and the bone is never involved.

Epithelioma.—In this affection the progress is slower, there is only one point of ulceration: the secretion is scanty, and the border is markedly infiltrated.

Bullous Syphiloderm.—This is a late manifestation, and is characterized by an eruption of well-filled blebs varying in size from a coffee-bean to a walnut. The contents of the blebs are puriform. They subsequently form dark, conic, stratified crusts under which are ulcers pouring forth a thick, purulent fluid.

Diagnosis.—*Pemphigus.*—The history, the concomitant symptoms of syphilis, and thick, greenish crusts will serve to distinguish syphilis from pemphigus.

Gummatous Syphiloderm.—This appears as a firm, circumscribed nodule that gradually turns red and softens. It may disappear by absorption, or break down and leave a deep, punched-out ulcer.

Moist Papules (Mucous Patches).—These consist in soft flat papules covered with an offensive, grayish secretion. Heat and moisture favor their development, so that their favorite seats are around the arms, the genitalia, the mouth, and in women under the mammæ.

Papulosquamous Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of small papules that are more or less scaly, so as to resemble psoriasis.

Diagnosis.—The history, the slight scaling, the dirty-gray color of the scales, the dark-red color of the lesions, the especial tendency to involve the palms and soles, will serve to distinguish syphilis from *psoriasis.*

Squamous Eczema.—In this affection the distribution, the infiltration of the skin, and the marked itching will lead to a correct diagnosis.

Annular Syphiloderm.—In this form the lesions consist of circles or semicircles of small, dark-red papules.

Pustular Syphiloderm.—This form usually appears within the first year, and is characterized by a general eruption of small or large, acuminated or flat pustules that finally dry up and form yellowish-brown crusts. Large lesions leave superficial ulcers. The term *rupia* is applied to large, conic, stratified crusts that rest loosely on the ulcerating basis.

Diagnosis.—*Variola.*—Absence of syphilitic history, the shot-like feel, the umbilication, the itching, the high fever, and the acute course will separate variola from syphilis.

Acne.—This is usually limited to the face and shoulders; there is no history of syphilis or concomitant symptoms of that affection.

Treatment.—The internal treatment consists in the administration of antisyphilitic remedies (salvarsan, mercury, and iodids) and general tonics.

℞. Hydrargyri iodidi	gr. j
Potassii iodidi	ʒiv
Syrupi sarsaparillæ compositi	
Aquæ	āā f℥ij.—M.

SIG.—Teaspoonful three times a day after meals.

(R. W. TAYLOR.)

Or:

℞. Hydrargyri protiodidi	gr. v–x
Extracti opii	gr. iv.—M.

Fiant pilulæ No. xx.

SIG.—One morning and evening. (HARDAWAY.)

Local Treatment.—Papular eruptions may be washed with mercurial lotions; mucous patches may be dusted with calomel; ulcers may be dressed with iodoform.

EPITHELIOMA

(Skin Cancer)

Etiology.—Late life, heredity, and local irritation are the predisposing factors.

Varieties.—Superficial, deep-seated, and papillomatous.

Superficial Epithelioma (Rodent Ulcer).—This form usually begins as a firm, circumscribed, reddish-yellow, wax-like papule. After the lapse of several months or years the papule becomes scaly, and the removal of the scales is followed by a slight excoriation, which in turn becomes covered with a slight, reddish-brown crust. The latter tends to adhere, and its repeated removal is followed by a raw surface, which is gradually converted into an ulcer. The ulcer has a prominent indurated margin; its outline is irregular; its base is uneven and glazed; and it exudes a sanious, viscid excretion. It is not painful; it does not lead to enlargement of the neighboring lymphatic glands; nor does it cause impairment of the general health. It spreads very slowly, and sometimes becomes stationary or actually heals. More frequently the ulceration continues until it involves all the tissues of the part, even the bones. The ulcer usually appears on the face, and in its advance it may destroy the nose, eyes, or a large proportion of the cranial bones.

Deep-seated Epithelioma.—This variety may begin as a deep-seated, red, shiny tubercle, or it may develop from the superficial form. The ulcer which is ultimately formed is deep; its base is granular; its edges are everted, indurated, and of a reddish-purple color; it secretes a blood-stained yellow fluid; it is the seat of lancinating pain; it causes enlargement of the neighboring glands; and it sooner or later induces the cancerous cachexia. Death may result from exhaustion, or, more rarely, from hemorrhage caused by ulceration of a large blood-vessel.

Papillomatous Epithelioma.—This may begin as a warty excrescence, or may develop from one of the preceding varieties. It is characterized by an ulcerated surface from which springs an aggregation of large, highly vascular papillæ. Between the papillæ there are often deep-seated fissures from which exudes an offensive viscid discharge. The general health is impaired and the neighboring glands are enlarged.

Diagnosis.—*Lupus Vulgaris.*—Lupus begins in the young; the original papule is soft; there is often more than one center of ulceration; *the margins of the* ulcer are not hard and everted;

the progress is extremely slow; the discharge from the ulcer is very scant, and the bones are never involved.

Syphilis.—The history, the associated evidences of syphilis, the rapid progress of the ulceration, the abundant discharge, the absence of pain, and the effect of treatment will suggest the diagnosis.

Prognosis.—This should be guarded in rodent ulcer and the deep-seated epitheliomas. A thorough removal in the beginning of the disease is often followed by a permanent cure. When the process is advanced, the growth usually returns.

Treatment.—Epitheliomatous growths may be removed by the use of caustics, the cautery, the curet, or by excision. The last is preferable if the growth is small, circumscribed, and in accessible region.

X-rays, radium and Finsen light often prove effective.

AINHUM

Ainhum is a rare affection, occurring chiefly in the colored race, and characterized by the appearance of a groove or furrow at the base of one or more of the toes. The groove deepens, the affected member becomes swollen, and finally drops off at the point of strangulation.

PRURITUS

Definition.—Pruritus is a functional affection, characterized by itching which is unassociated with any objective phenomena.

Etiology.—Pruritus may arise without obvious cause, as the *pruritus senilis* observed in the old, and the *pruritus hiemalis* which develops on the approach of cold weather and disappears when the weather becomes warm.

Symptomatic Pruritus.—Pruritus may be a symptom of many conditions, notably diabetes, gout, hysteria, neurasthenia, and Bright's disease.

Symptoms.—There is only one symptom, and that is itching; but as a result of scratching, the part may become hyperemic, thickened, or the seat of eczema.

Diagnosis.—Pruritus must be distinguished from the itching induced by *pediculosis, or some local disease, like eczema.*

Prognosis.—This will depend upon the cause. If the primary disease is curable, the prognosis for permanent relief is favorable. In other cases temporary relief only is to be expected.

Treatment.—Search should be made for the exciting cause, which should be removed, if possible. In all cases the urine must be examined for sugar, since diabetes is one of the most frequent causes of pruritus. Among the internal remedies recommended for pruritus may be mentioned nux vomica, belladonna, and pilocarpin. The best local remedies are carbolic acid, vinegar, thymol, chloral-camphor, boric acid resorcin, menthol, hydrocyanic acid, and hot water.

℞.	Resorcinolis		gr. xv–xxx
	Sodii chloridi		gr. xv
	Glycerini		fʒij
	Liquoris calcis	q. s. ad	f℥iv.—M.

(HARTZELL.)

℞.	Acidi carbolici		ʒj–iij
	Glycerini		fʒij
	Alcoholis		f℥ij
	Aquæ	q. s. ad	Oj.—M.

℞.	Acidi carbolici	gr. xv
	Hydrargyri chloridi mitis	gr. xx
	Unguenti zinci oxidi	℥j.

SIG.—Apply locally in pruritus ani.

RINGWORM

Definition.—A contagious disease excited by a vegetable parasite—the small-spore fungus (*Microsporum*) or the large-spore fungus (*Tricophyton*).

Varieties.—On the scalp it is termed *tinea tonsurans;* on the body, *tinea circinata;* on the bearded region, *tinea sycosis.*

TINEA TONSURANS

This form is observed almost exclusively on the scalp of children. It is characterized by one or more rounded, scaly, elevated, grayish-colored patches through which project dry, brittle, lusterless, broken-off hairs.

Diagnosis.—*Seborrhea.*—The patches are not circumscribed; the scales are greasy; the hair is not involved; and the microscope reveals no parasite.

Eczema.—The patches are not circumscribed; the hair is not involved; there is more inflammation; there is marked itching; and the microscope reveals no parasite.

Alopecia Areata.—Baldness is complete; there are no scales; and the base is smooth and shiny.

Prognosis.—The disease is stubborn, but usually yields to energetic treatment in the course of several months.

Treatment.—Tonics are often indicated. The parts should be thoroughly washed with soap and water, and the affected hairs removed. The following parasiticides may be employed in ointment or lotion: mercury, betanaphthol, sulphur, chrysarobin, or sulphurous acid.

℞. Hydrargyri ammoniati	ʒj
Petrolati	ʒj.

SIG.—Apply once or twice daily.

Or:

℞. Betanaphtholis	gr. xl
Sulphuris præcipitati	ʒj
Vaselini	℥j.—M.

SIG.—Rub into affected area once or twice daily.

(HARDAWAY.)

Treatment by *x*-rays is commonly very effective.

TINEA CIRCINATA

(Ringworm of the Body)

This appears as one or more rounded, red, slightly elevated scaly patches, which on close examination reveal minute vesicles or papules. As the disease advances new patches spring from the periphery, while the central portion clears up. There is often considerable itching.

Diagnosis.—*Psoriasis.*—The marked scaling; the absence of itching; the tendency to involve the extensor surfaces, especially the knees and elbows; and the absence of the fungus will separate psoriasis from ringworm.

Eczema.—The patches are ill defined; do not clear in the center; there is more infiltration of the skin; and there is no parasitic fungus.

Prognosis.—The disease usually yields rapidly to treatment.

Treatment.—Mercury, sulphur, sulphurous acid, and hyposulphite of sodium are among the best parasiticides.

℞. Sodii hyposulphitis	ʒij
Aquæ	f℥ij.—M.
Sig.—Apply locally.	(Duhring.)

Or:

℞. Hydrargyri ammoniati	gr. xxx
Adipis	℥j.—M.
Sig.—Apply locally.	

TINEA SYCOSIS

(Barber's Itch; Sycosis Parasitica)

This begins as a red, scaly patch involving the bearded region. Soon purplish tubercles and pustules form around the opening of the hair-follicles, and the hairs become lusterless, brittle, and loose. There is often considerable itching.

Diagnosis.—*Simple Sycosis.*—In this the inflammation is superficial; the hairs are not involved; and the trichophyton is absent.

Eczema.—The tubercles, the involvement of the hairs, and the presence of the trichophyton will separate it from eczema.

Prognosis.—Recovery usually ensues in the course of a few weeks.

Treatment.—The affected hairs should be removed, and one of the following parasiticides employed in lotion or ointment: mercury, sulphur, or hyposulphite of sodium.

℞. Sodii hyposulphitis	ʒiij
Aquæ	f℥iij.—M.

Sɪɢ.—Apply locally.

Or:

℞. Sulphuris sublimati	ʒij
Vaselini	℥ij.—M.

Sɪɢ.—Apply locally.

TINEA VERSICOLOR

(Pityriasis Versicolor)

Definition.—A chronic affection excited by a vegetable parasite, the *Microsporon furfur*, and characterized by fawn-colored scaly patches which usually appear about the chest.

Etiology.—It is a disease of adult life, and is mildly contagious.

Symptoms.—It appears usually on the front of the chest as small round spots of a pale-yellow or fawn color, which slowly enlarge, fuse, and form slightly elevated, scaly patches. Subjective symptoms are usually absent.

Diagnosis.—*Chloasma* somewhat resembles tinea versicolor, but the former is not often observed on the trunk, is not scaly, and is not associated with a parasite.

Prognosis.—The disease is readily curable, but relapse is not uncommon.

Treatment.—The parts should be frequently washed with soap and water, after which one of the following parasiticides may be applied: Corrosive sublimate (2–3 grains to an ounce of water), sulphurous acid, or hyposulphite of sodium:

℞. Sodii hyposulphitis	ʒv
Glycerini	fʒiij
Aquæ q. s. ad	f℥v.—M.

Sɪɢ.—Apply locally.

Or:

℞. Hydrargyri chloridi corrosivi	℈j
Alcoholis	f℥iv
Saponis viridis	℥ij
Olei lavandulæ	fʒj.—M.

Sɪɢ.—To be rubbed in well night and morning.

(Van Harlingen.)

FAVUS

(Tinea Favosa)

Definition.—A contagious affection of the scalp excited b the Achorion Schönleinii, and characterized by yellowis cup-shaped crusts.

Etiology.—It is observed especially in poor, ill-nourishe children. It is rare in America.

Symptoms.—The disease is characterized by one or mo rounded, yellow, cup-shaped crusts, through which proje dry, brittle, lusterless hairs. The underlying tissue is mo or less atrophied and scarred. It is associated with som itching and a peculiar musty odor.

Diagnosis.—The yellow, cup-shaped crusts, the odor, an the atrophy of the skin will separate it from *ringworm*.

Prognosis.—Favus is very resistant to treatment and not rarely followed by permanent areas of baldness.

Treatment.—The crusts should be removed by oil or soa and water. The affected hairs should also be removed. Th following parasiticides are efficient: mercury, sulphur, chrys robin, and hyposulphite of sodium. *X*-ray treatment giv good results.

SCABIES

(Itch)

Definition.—Scabies is a contagious disease excited by a animal parasite—the Acarus scabiei—and manifested b papules, vesicles, pustules, burrows, and intense itching.

Etiology.—The disease is always acquired through intimate intercourse with patients already affected.

Symptoms.—The disease manifests itself by intense itching, which is associated with an eruption of small papules, vesicles, and pustules. Among these lesions may be found cuniculi, or burrows (zig-zag tracks); these are discolored, dotted, slightly elevated lines, ranging from a line to half an inch in length, and produced by the penetration of the female acarus and the deposition of her eggs along the passage. The parts most commonly affected are the hands, between the fingers, the wrists, the axillæ, the genitalia, beneath the mammæ, and the inner aspects of the thighs. The face and scalp are never involved.

Diagnosis.—The recognition of scabies rests on the history, the itching, the presence of burrows, the multiformity of the lesions, and their peculiar distribution.

Prognosis.—The disease is, as a rule, readily cured.

Treatment.—Ointments of sulphur, styrax, and naphthol are efficient remedies. After a thorough bath the whole body should be anointed twice daily for three or four days. At the end of this time the bath should be repeated, and the bed-linen and underclothing changed. The infected clothing should be sterilized.

℞. Sulphuris sublimati	ʒj
Balsami Peruviani	ʒss
Adipis	℥j.—M.

SIG.—Rub in thoroughly twice daily. (DUHRING).

℞. Balsami styracis	ʒiv
Adipis	℥iss.—M.

PEDICULOSIS

(Phtheiriasis)

Pediculosis Capitis.—This form results from the pediculus capitis, or head-louse, a gray insect from one to two millimeters in length. The condition is recognized by itching of the scalp and the discovery of the lice or their white ova, or nits. Eczematous lesions resulting from scratching are often observed.

Pediculosis Corporis.—This form results from the pediculus corporis, pediculus vestimenti, or body-louse, a somewhat larger insect than the head-louse. The condition is recognized by intense itching on the covered parts of the body, scratch-marks, petechiæ caused by the bite of the insect, and the discovery of the lice on the garments.

Pediculosis Pubis.—This form results from the pediculus pubis, or crab-louse, a minute, gray, translucent insect. It is found on parts covered with short hair, as the pubes, axillæ, eyebrows, etc.

Treatment.—In *pediculosis capitis* the head may be thoroughly washed with coal-oil, dilute carbolic acid (1 dram to 1 pint), or ointment of ammoniated mercury (20 grains to the ounce), or tincture of cocculus indicus.

In *pediculosis corporis* the parts should be thoroughly washed and the clothes subjected to a high temperature. The body may be bathed in a weak solution of corrosive sublimate.

In *pediculosis pubis* a lotion of corrosive sublimate (2 grains to 1 ounce) or an ointment of ammoniated mercury (1 dram to 1 ounce) is very efficient.

INDEX

ABDOMINAL aorta, aneurysm of, 233
  distention, 27
Abiotrophy, 480
Abscess of brain, 472
  of liver, 109
  of lung, 295
  retropharyngeal, 38
Acarus scabiei, 611
Acetonuria, 132
Achondroplasia, 185
Achorion schönleinii, 611
Achylia gastrica, 25, 44
Acid, butyric, test for, 23
  free, test for, 21
  HCL, test for, qualitative, 22
    quantitative, 22
  lactic, test for, 23
Acidity of gastric contents, 25
Acidosis, 132
Acne atrophica, 566
  hypertrophicæ, 566
  indurata, 566
  papulosa, 566
  pustulosa, 566
  rosacea, 568
  vulgaris, 565
Acromegaly, 185
Adams-Stokes syndrome, 201
Addison's disease, 180
  keloid, 595
Adiposis dolorosa, 187
Agraphia, 473
Ague, fever and, 339
Ainhum, 606
Albinism, 585
Albuminuria, 131
Albumosuria, 132
Alcoholism, acute, 534
  chronic, 535
Alkaptonuria, 124
Alopecia, 586
  areata, 587
Amebic dysentery, 394
Amyloid degeneration of kidney, 148
  liver, 113
Amyotonia, congenital, 528
Amyotrophic lateral sclerosis, 482
Anacidity of gastric contents, 25
Analgesia, 441
Anemia, 166
  aplastic, 169
  cerebral, 460
    headache of, 499
  pernicious, 168
  primary, 167, 170
  primitive splenomegaly with, 189
  secondary, 167
  splenic, 189
Anesthesia, 440
  dissociative, 441
Aneurysm of abdominal aorta, 233
  of aorta, 231
  thoracic, 231
Angina, Ludwig's, 37
  pectoris, 226
  simple, 35
  Vincent's, 36
Angioma, 598
  cavernosum, 598
  simplex, 598
Angioneurotic edema, 533
Anhydremia, 163
Anisocoria, 446
Ankle-clonus, 439
Ankylostomiasis, 412
Ankylostomum duodenale, 412
Anorexia, 19
Anuria, 122
Aorta, aneurysm of, 231
Aortic insufficiency, 214
  stenosis, 214
Apex-beat, 191
  changes in force and extent of, 192
  displacement of, 192
Aphasia, 473
Aphonia, 236
Aphthous stomatitis, 29
Apoplexy, cerebral, 461
  pulmonary, 279

Appendicitis, 79
  varieties of, 80
Appetite, 19
  inordinate, 19
  loss of, 19
Argyll-Robertson pupil, 446, 457
Arhythmia, 198
Arm-jerk, 439
Arteries, cerebral, obstruction of, 466
  diseases of, 229
Arteriosclerosis, 229
Arteritis, chronic, 229
Arthritis deformans, 528
  monarticular, 530
  rheumatoid, 528
Arthropathies, 443
Ascaris lumbricoides, 411
Ascites, 119
Asterognosis, 441
Asthenic bulbar palsy, 528
Asthma, 270
  bronchial, 270
  hay, 274
Ataxia, Friedreich's, 490
  hereditary, 490
  locomotor, 485
  Marie's, 491
Ataxic gait, 437
  paraplegia, 489
Ateliosis, 187
Atheroma, 229
Athetosis, 437
Atony of stomach, 47
Atrophy, muscular, 442
  of nails, 541
  of optic nerve, 447
  progressive spinal muscular, 480
Auricular fibrillation, 200
Auscultation of heart, 195
  of lungs, 246

Babinski's reflex, 439, 463
Bacillary dysentery, 391
Baldness, 586
Banti's disease, 189
Barbadoes leg, 595
Barber's itch, 569, 609
Barlow's disease, 427
Barrel-chest, 35
Basedow's disease, 181
Bednar's aphthæ, 29
Bell's mania, 448
  palsy, 497
Bence-Jones' protein, 132
Beriberi, 428
Bile-ducts, catarrh of, 95
Biliary calculi, 98
Black tongue, 18
Blackheads, 558
Blebs, 546
Bleeding gums, 17
  occult, 26
Blood, alkalinity of, 158
  diseases of, 157
  drying and staining of, 161
  examination of, 157
  normal, 157
  parasites in, 165
  specific gravity of, 158
Blood-corpuscles, enumeration of red, 159
    of white, 160
  study of white, 160
Blood-forming organs, diseases of, 157
Blood-pressure, 203
Blue line on gums, 17
Bluish-black discoloration of tongue, 18
Boas and Ewald, test-breakfast of, 21
Boas' resorcin-sugar test, 22
Boil, 570
Böttger's test for glycosuria, 130
Bradycardia, 198
Brain, abscess of, 472
  congestion of, 459
  disease, organic, headache of, 499
  diseases of, 450
  morbid growths in, 467
  tumors of, 467
  water on, 454
Break-bone fever, 400
Breath, fetor of, 19
  sounds, 246
Bright's disease, acute, 141
Brill's disease, 332
Broadbent's sign, 207
Bromidrosis, 555
Bronchi, dilatation of, 268
  diseases of, 262
Bronchial breathing, 246
Bronchiectasis, 268
Bronchitis, acute catarrhal, 262
  capillary, 289
  chronic, 265
  fibrinous, 267
Bronchophony, 248

Bronchopneumonia, 289
Bronchorrhagia, 278
Brown-Séquard's paralysis, 432
Bruit, aneurysmal, 197
Bulbar palsy, asthenic, 528
  paralysis, 483
Bulimia, 19
Bullæ, 546
Burns, 579
Butyric acid, test for, 23

CACHEXIA, malarial, 344
Caisson disease, 492
Calculi, biliary, 98
  pancreatic, 90
  renal, 152
Callositas, 591
Callus, 591
Cancer of liver, 110
  of pancreas, 88
  of skin, 604
  of stomach, 59
Cancrum oris, 30
Capillary nevus, 598
Carbohydrates, test for, 24
Carbunculus, 570
Catalepsy, 445
Catarrh, acute gastric, 40
  autumnal, 274
  chronic bronchial, 265
    gastric, 43
    nasal, 252
  epidemic, 377
  of bile-ducts, 95
Catarrhal dyspepsia, 43
Cephalalgia, 499
Cerebral anemia, 460
    headache of, 499
  apoplexy, 461
  arteries, obstruction of, 466
  hemorrhage, 461
  hyperemia, 459
    headache of, 499
  leptomeningitis, acute, 450
    chronic, 452
  pachymeningitis, chronic, 453
  paralysis in children, 458
Cerebrospinal fever, 336
  meningitis, epidemic, 336
  sclerosis, disseminated, 489
Cervical pachymeningitis, hypertrophic, 476
Cestode infection, 409
Chapping, 574
Charcot-Leyden crystals, 239
Charcot's hepatic fever, 100
  joints, 487
Chest, mensuration of, 250
  radioscopy of, 250
Cheyne-Stokes breathing, 237
Chicken-pox, 361
Child-crowing, 259
Childhood, diarrhea in, 73
Children, cerebral paralysis in, 458
Chills and fever, 339
Chloasma, 589
Chlorids in urine, 127
Chlorosis, 170
Cholangitis, catarrhal, 95
  suppurative, 97
Cholecystitis, acute, 97
Cholelithiasis, 98
Cholemia, 94
Cholera, 387
  infantum, 74
  morbus, 73
  nostras, 73
Choluria, 134
Chorea, acute, 514
  Huntingdon's, 515
  insaniens, 515
  minor, 514
  Sydenham's, 514
  varieties of, 436
Choreiform movements, 435
Chromidrosis, 555
Chvostek's sign, 188
Chyluria, 135
Circulatory system, diseases of, 191
Cirrhosis, alcoholic, 104
  atrophic, 104
  capsular, 108
  Hanot's hypertrophic, 107
  hypertrophic biliary, 107
  Laennec's, 104
  of liver, 103
    syphilitic, 108
  of lung, 294
  of pancreas, 88
Claudication, intermittent, 230
Clavus, 501, 591
Claw-hand, 476
Coffee-ground vomit, 57
Cold in the head, 251
Colic, intestinal, 70
  biliary, 99
  pancreatic, 90

Colic, renal, 152
Colitis, mucous, 78
Coma, 444
  diabetic, 422
  uremic, 140
Comedo, 558
Compensation in valvular disease of heart, 213
Concato's disease, 108
Congenital amyotonia, 528
  internal hydrocephalus, 454
  myatonia, 528
  myotonia, 527
Congestion of brain, 459
Consciousness, disturbances of, 444
Constipation, habitual, 67
Consumption, pulmonary, 297
Convulsions, 433
  general clonic, 434
  hysteric, 434
  local, 435
  tonic, 434
Corn, 591
  cutaneous, 592
  soft, 592
Cornu cutaneum, 592
Corrigan's pulse, 202
Coryza, 251
Cough, 237
  winter, 265
Courvoisier's law, 100
Cow-pox, 360
Cracked-pot sound, 245
Craniotabes, 420
Cretinism, 185
Croup, 256
  membranous, 257
Crusts, 550
Curschmann's spirals, 271
Cyanosis, general, 204
Cylindruria, 127
Cystinuria, 124
Cysts of pancreas, 89

Dalrymple's Sign, 182
Dandruff, 556
Dandy fever, 400
Deafness, 447
Decompensation, period of, in valvular heart disease, 218
Decubitus, 443
Degeneration, reaction of, 443
Delirium, 448
  cordis, 199
Delirium tremens, 535
Delusions, 447
Dementia, paretic, 455
Demodex folliculorum, 558
Dengue, 400
Dentition, delayed, 17
Dercum's disease, 187
Dermatitis, 578
  calorica, 579
  contusiformis, 561
  exfoliativa, 581
  herpetiformis, 577
  medicamentosa, 580
  traumatica, 578
  venenata, 579
Diabetes insipidus, 425
  mellitus, 421
  phosphaticus, 127
  renal, 129
Diarrhea, 71
  in childhood, 73
Diazo-reaction, Ehrlich's, 135
Dibothriocephalus latus, 409
Dicrotic pulse, 202
Dietl's crisis, 138
Digestive system, 17
Dilatation of bronchi, 268
  of stomach, 61
    acute, 65
Diphtheria, 362
Discoloration of tongue, 18
Distention, abdominal, 27
Divers' paralysis, 492
Diverticula of esophagus, 40
Dizziness, 503
Dobell's solution, 38
Dropsy, 204
Drug eruptions, 580
Ductless glands, diseases of, 180
Duhring's disease, 577
Dum-dum fever, 407
Duodenum, ulcer of, peptic, 54
    perforating, 54
    round, 54
Dura mater, hematoma of, 453
Durosiez's sign, 215
Dysentery, 391
  amebic, 394
  bacillary, 391
Dysidrosis, 585
Dyspepsia, catarrhal, 43
  nervous, 44, 48
Dysphagia, 20
Dyspnea, 236

Dystrophy, Erb's juvenile, 527
  Landouzy-Dejerine type, 527
  muscular, 526

Ear, diseases of, 447
Ebstein's sign, 207
Ecchymoses, 543
Echinococcus of liver, 112
Eclampsia, 435
Ecthyma, 581
Eczema, 573
  erythematosum, 573
  fissum, 574
  impetiginosum, 574
  madidans, 574
  papulosum, 574
  pustulosum, 574
  rubrum, 574
  squamosum, 574
  verrucosum, 574
  vesiculosum, 574
Edema, angioneurotic, 533
  of glottis, 260
  of larynx, 260
  of lungs, 282
Eel-worm, 411
Egophony, 248
Ehrlich's diazo-reaction, 135
Electromuscular contractility, changes in, 442
Elephantiasis, 595
  græcorum, 402
Embolism, cerebral, 466
  of mesenteric vessels, 121
Embryocardia, 196
Emesis, 20
Emphysema, hypertrophic, 275
  of skin, 541
  pulmonary, 275
Empyema, 309, 312
Encephalitis, suppurative, 472
Endocarditis, 209
  chronic, 210, 212
  infective, 212
Enlargement of tongue, 18
Enteralgia, 70
Enteric fever, 322
Enteritis, acute, 72
  chronic, 77
Enteroptosis, 65
Enterorrhagia, 26
Eosinophilia, 165
Epilepsy, 504
  Jacksonian, 506
Epistaxis, 235
Epithelioma, 604
  deep-seated, 605
  papillomatous, 605
  superficial, 604
Erb's juvenile dystrophy, 527
  sign, 188
Eruptions from drugs, 580
  of skin, 542
Erysipelas, 371
Erythema, diffuse, 544
  intertrigo, 560
  multiforme, 561
  nodosum, 561
  scarlatinoides, 581
  simplex, 560
Erythremia, 178
Esophageal obstruction, organic, 39
Esophagismus, 39
Esophagus, diverticula of, 40
  spasm of, 39
  stenosis of, 39
Estivo-autumnal fever, 343
Ewald and Boas, test-breakfast of, 21
Exhaustion, heat, 534
Exophthalmic goiter, 181
Expectoration, 238
Extra-systoles, 199
Eye, conjugate deviations of, 446
  diseases of, 446

Facial paralysis, 497
Famine fever, 334
Favus, 611
Febricula, 321
Fehling's test for glycosuria, 129
Fermentation test for glycosuria, 130
Ferret eyes, 333
Festination, 438
Fetor of breath, 19
Fever, 316
  break-bone, 400
  catarrhal, 377
  causes of, 318
  cerebrospinal, 336
  chills and, 339
  continued, 343
  dandy, 400
  degree of, 317
  detection of, 316
  dum-dum, 407
  effects on tissues, 319
  enteric, 322
  ephemeral, 321

- Fever, estivo-autumnal, 343
  - famine, 334
  - glandular, 406
  - hepatic, 100
  - incubation period of, 319
  - jail, 332
  - malarial, 339
    - intermittent, 342
    - pernicious, 343
  - Malta, 407
  - Mediterranean, 407
  - protracted cases of, 320
  - relapsing, 334
  - remissions of, 317
  - remittent, 343
  - rheumatic, 367
  - Rocky Mountain spotted, 406
  - scarlet, 347
  - ship, 332
  - simple continued, 321
  - spirillum, 334
  - spotted, 336
  - stages of, 316
  - symptoms of, 318
  - terminations of, 317
  - thermic, 533
  - tick, 334
  - types of, 317
  - typhoid, 322
  - typhus, 332
  - yellow, 380
- Fever-blisters, 564
- Fevers, jaundice in, 321
  - protection from future attacks of, 320
- Fibrillation, auricular, 200
- Fibroma molluscum, 597
- Filaria bancrofti, 414
  - diurna, 414
  - loa, 414
  - sanguinis hominis, 414
- Fingers, clubbed, 542
- Fish-skin disease, 594
- Fleischl's hemoglobinometer, 157
- Floating kidney, 137
- Folliculitis barbæ, 568
- Foot, perforating ulcer of, 444
- Freckle, 588
- Fremitus, vocal, 243
- Friction-sounds, 197, 249
- Friedreich's ataxia, 436, 490
  - sign, 207
- *Frölich*'s syndrome, 186
- *Frost*-bite, 579
- Functional capacity of kidney
  - tests of, 136
- Funnel breast, 241
- Fur on tongue, 17, 18
- Furunculus, 570

- GAIT, ataxic, 437
  - of pseudomuscular hypertroph 438
  - spastic, 437
  - steppage, 438
- Gall-stones, 98
- Gangrene, symmetric, 444, 532
- Gastralgia, 52
- Gastrectasis, 61
- Gastric catarrh, acute, 40
    - chronic, 43
  - contents, acidity of, 25
      - normal, 25
    - anacidity of, 25
    - examination of, 21
    - hyperacidity of, 25, 50
    - subacidity of, 25
    - superacidity of, 50
- Gastritis, acute, 40
  - chronic, 43
- Gastrodynia, 52
- Gastroptosis, 65
- Gastrorrhagia, 66
- Gastrosuccorrhea, 51
- Gaucher's disease, 189
- German measles, 354
- Giddiness, 503
- Gin-drinker's liver, 104
- Girdle sensation, 442
- Glandular fever, 406
- Glénard's disease, 65
- Glossolabiolaryngeal paralysis 483
- Glottis, edema of, 260
  - spasm of, 259
- Glycosuria, 128
  - qualitative tests for, 130
  - quantitative tests for, 130
- Gmelin's test for choluria, 134
- Goiter, exophthalmic, 181
- Gout, 415
  - varieties of, 416
- Grand mal, 506
- Gravel in kidney, 152
- Graves' disease, 181
- Green sickness, 170
- Grocco's sign, 310
- Gums, 17

- Günzburg's phloroglucin-vanillin test, 22

- HABITUAL constipation, 67
- Hair, trophic affections of, 444
- Hallucination, 448
- Hanot's hypertrophic cirrhosis, 107
- Harrison's groove, 420
- Hay fever, 274
- Hay's test for choluria, 135
- Head, swimming in, 503
- Headache, 499
  - hysteric, 500
  - indurative, 501
  - neurasthenic, 500
  - of cerebral anemia, 499
    - hyperemia, 499
  - of organic brain disease, 499
  - reflex, 500
  - sick-, 520
  - toxemic, 500
- Hearing, hyperacusis of, 447
- Heart, apex-beat of, 191
  - auscultation of, 195
  - chronic valvular disease of, 213
  - dilatation of, 221
  - diseases of, 205
  - enlargement of, 221
  - fatty degeneration of, 224
    - infiltration of, 224
  - fibroid induration of, 225
  - hypertrophy of, 221
  - neuralgia of, 226
  - palpation of, 194
  - palpitation of, 204
  - percussion of, 194
- Heart-block, 200
- Heart-sounds, adventitious, 196
  - alteration in rhythm of, 196
  - intensity of, 195
- Heat exhaustion, 534
  - stroke, 533
- Heberden's nodes, 530
- Heller's test for albuminuria, 131
    - for hematuria, 133
- Helminthiasis, 409
- Hematemesis, 21, 66
- Hematoma of dura mater, 453
- Hematoporphyrinuria, 128
- Hematuria, 133
- Hemianesthesia, 440
- Hemicrania, 520
- Hemiplegia, 431
- Hemoglobin, estimation of, 157
- Hemoglobinometer, Tallqvist's, 157
- Hemoglobinuria, 134
- Hemopericardium, 209
- Hemophilia, 175
- Hemoptysis, 278
- Hemorrhage, bronchopulmonary, 278
  - cerebral, 461
- Hemothorax, 315
- Henoch's electric chorea, 436
  - purpura, 177
- Hepatitis, acute parenchymatous, 114
    - suppurative, 109
  - chronic interstitial, 103
- Hereditary ataxia, 490
- Herpes facialis, 564
  - gestationis, 577
  - progenitalis, 564
  - simplex, 564
  - zoster, 565
- Hiccup, 26
- Hirsuties, 594
- Hives, 562
- Hodgkin's disease, 174
- Hookworms 412
- Huntingdon's chorea, 436, 515
- Hutchinson's teeth, 17
- Hydatid cyst of liver, 112
- Hydremia, 163
- Hydrocephalus, chronic, 454
- Hydronephrosis, 154
- Hydropericardium, 209
- Hydrophobia, 401
- Hydrothorax, 313
- Hypopituitarism, 186
- Hymenolepis nanus, 410
- Hyperacidity of gastric contents, 25, 50
- Hyperacusis of hearing, 447
- Hyperchlorhydria, 25, 44, 50
- Hyperemia, cerebral, 459
    - headache of, 499
  - of kidneys, 138
  - of liver, 102
- Hyperesthesia, 441
- Hyperidrosis, 554
- Hyperpiesis, 229
- Hyperthyroidism, 181
- Hypertrichosis, 594
- Hypertrophic cervical pachymeningitis, 476
- Hypertrophy of tonsils, 34
  - pseudomuscular, 526

Hypochylia gastrica, 25
Hypoleukocytosis, 165
Hypothyroidism, 183
Hysteria, 436, 508
Hysteric headache, 500

ICHTHYOSIS, 594
Icterus, 92
  gravis, 114
  neonatorum, 94
Idiopathic infantilism, 187
Ileocolitis, 76
Illusion, 448
Imperative conception, 448
Impetigo contagiosa, 583
Indicanuria, 134
Indurative headache, 501
Inebriety, 534
Infantilism, idiopathic, 187
Infections, pyogenic, 374
Infectious diseases, acute, 316
Inflammation of mouth, 28
Influenza, 377
Insane, general paralysis of, 455
Insolation, 533
Insufficiency, aortic, 214
  mitral, 217
  pulmonary, 218
  tricuspid, 217
Insular sclerosis, 489
Intercostal neuralgia, 517
Interstitial nephritis, chronic, 146
Intestinal colic, 70
Itch, 611

JACKSONIAN EPILEPSY, 506
Jail fever, 332
Jaundice, 92
  acholuric, 95
  catarrhal, 95
    chronic, 96
  in fevers, 321
Jaw-jerk, 439
Johnson's test for albuminuria, 131
Joints, diseases of, 524

KAKKE, 428
Kala-azar, 407
Keloid, 596
  Addison's, 595
Keratoma, 591
Keratosis pilaris, 589
Kernig's sign, 439
Ketonuria, 132
Kidney, amyloid degeneratio
    148
  contracted, 146
    white, 144
  cyanotic induration of, 139
  floating, 137
  gouty, 146
  lardaceous, 148
  large white, 144
  malignant tumors of, 156
  movable, 137
  polycystic disease of, 156
  red granular, 146
  tuberculosis of, 155
  waxy, 148
Kidneys, diseases of, 122
  hyperemia of, 138
  tests of functional capacity
    136
Kleptomania, 448
Knee-jerks, 438
Koplik's sign, 353
Korsakow's syndrome, 495
Kussmaul's sign, 193

LACTIC ACID, test for, 23
Laennec's cirrhosis, 104
La grippe, 377
Landouzy-Dejerine dystrophy,
Landry's disease, 479, 484
Lardaceous kidney, 148
  liver, 113
Laryngismus stridulus, 259
Laryngitis, acute catarrhal, 25
  chronic, 257
  syphilitic, 258
  tuberculous, 258
Laryngospasmus, 259
Larynx, 235
  diseases of, 251
  edema of, 260
Lead-poisoning, chronic, 537
Legal's test for acetonuria, 132
Lentigo, 588
Lepra, 402
Leprosy, 402
Leptomeningitis, cerebral, a
    450
    chronic, 452
  spinal, 474
Leucinuria, 124
Leukemia, 171
Leukocythemia, 171
Leukocytosis, 164

Motion, disturbances of, 430
Motor power of stomach, 24
Mouth, inflammation of, 28
Movable kidney, 137
Mucous colitis, 78
Mulberry rash, 333
Mumps, 379
Murmurs, cardiac, 196
Muscle sense, 442
Muscles, diseases of, 524
Muscular atrophy, 442
    progressive spinal, 480
  dystrophy, 526
  rheumatism, 524
Myalgia, 524
Myasthenia gastrica, 47
  gravis, 528
Myatonia, congenital, 528
Mycotic stomatitis, 29
Mydriasis, 446
Myelitis, 476
Myocardial disease, chronic, 224
Myocarditis, acute, 223
Myosis, 446
Myotonia, congenital, 527
Myxedema, 183

NÆVUS PIGMENTOSUS, 593
  vasculosus, 598
Nails, atrophy of, 541
  curving of, 542
  trophic affections of, 444
Nasal catarrh, chronic, 252
Negri bodies, 401
Nematode infection, 411
Nephritis, acute, 141
  chronic interstitial, 146
    parenchymatous, 144
Nephrolithiasis, 152
Nephroptosis, 137
Nerves, diseases of, 450
Nervous diseases, functional, 499
  prostration, 512
  system, diseases of, 430
Nettlerash, 562
Neuralgia, 442, 516
  intercostal, 517
  occipital, 517
  of heart, 226
  of stomach, 52
  trifacial, 517
Neurasthenia, 512
  gastrica, 48
Neurasthenic headache, 500
Neuritis, localized, 493
  multiple, 494
    endemic, 428
  optic, 447
Neuroses, occupation, 523
Nevus, capillary, 598
Nigrities linguæ, 18
Nodes, Heberden's, 530
Noma, 30
Nose, 235
  diseases of, 251
Nucleated red cells, 164
Nutrition, disturbances of, 442
  perverted, ulceration from, 443
Nystagmus, 446

OCCIPITAL NEURALGIA, 517
Occult bleeding, 26
Occupation neuroses, 523
Ochronosis, 125
Oligochromemia, 166
Oligocythemia, 166
Oliguria, 122
Oliver's sign, 232
Onychia, 542
Opium poisoning, chronic, 537
Oppenheim's disease, 528
Optic nerve, atrophy of, 447
Osmidrosis, 555
Osteitis deformans, 186
  Paget's, 186
Osteo-arthritis, 528
Oxaluria, 127
Oxyuris vermicularis, 411
Ozena, 253

PACHYMENINGITIS, chronic cerebral, 453
  hemorrhagic, 453
  hypertrophic cervical, 476
Paget's osteitis, 186
Palpation of liver, 91
  of lungs, 243
Palpitation of heart, 204
Palsy, asthenic bulbar, 528
  Bell's, 497
  shaking, 521
  writer's, 523
Paludism, 339
Pancreas, cancer of, 88
  cirrhosis of, 88
  cysts of, 89
  diseases of, 86
Pancreatic calculi, 90

Pancreatitis, acute, 86
  chronic, 88
Papillitis, optic, 447
Papules, 548
  moist, 603
Paralysis, 430
  acute ascending, 484
  infantile spiral, 397
  bulbar, 483
  divers', 492
  facial, 497
  glossolabiolaryngeal, 483
  in children, cerebral, 458
  irregular, 430
  of insane, general, 455
Paramyoclonus multiplex, 436
Paranesthesia, 441
Paraplegia, 432
  ataxic, 489
  primary spastic, 481
Parasites in blood, 165
Parenchymatous nephritis, acute, 141
    chronic, 144
Paresis, general, 455
Paresthesia, 441
Paretic dementia, 455
Parkinson's disease, 521
Parorexia, 19
Parotitis, 379
Parry's disease, 181
Patches, mucous, 603
Pectoriloquy, 248
Pediculosis capitis, 612
  corporis, 613
  pubis, 613
Peliosis rheumatica, 177
Pellagra, 539
Pemphigus, 582
  acutus, 582
  foliaceus, 583
  vulgaris, 582
Pepsin, test for, 24
Peptic ulcer, 44
    of duodenum, 54
    of stomach, 54
Percussion of heart, 194
  of liver, 92
  of lungs, 244
Perforating ulcer of duodenum, 54
    of foot, 444
    of stomach, 54
Pericarditis, 205
  purulent, 207
Pericardium, adherent, 207
Perihepatitis, chronic, 108
Peritonitis, acute, 115
  chronic diffuse, 118
Perityphlitis, 79
Pernicious anemia, 168
Pernio, 579
Pertussis, 384
Petechiæ, 543
Petit mal, 506
Pettenkofer's test for choluria, 135
Pharyngitis, acute, 35
  chronic, 37
Phenylhydrazin test for glycosuria, 130
Phosphaturia, 125
Phtheiriasis, 612
Phthisis, 297
Pica, 19
Pick's disease, 206
Pigeon-breast, 35
Pin-worm, 411
Pistol-shot sound, 215
Pityriasis rubra, 581
  versicolor, 610
Plague, 404
Plethora, 163
Pleura, diseases of, 308
Pleurisy, 308
  diaphragmatic 311
  purulent, 312
Pleuritis, 308
Pleurodynia, 524
Plumbism, 537
Pneumonia, catarrhal, 289
  central, 286
  chronic, 294
    interstitial, 294
  croupous, 283
  in children, 286
  lobar, 283
  lobular, 289
  massive, 286
  migratory, 286
  of drunkards, 286
  senile, 286
  stages of, 284
  typhoid, 286
Pneumonitis, 283
Pneumopericardium, 209
Pneumothorax, 314
Podagra, 415
Poikilocytosis, 164
Poisoning, chronic lead-, 537

Poisoning, opium, 537
Poker-spine, 530
Poliomyelitis, acute anterior, 397
    chronic anterior, 480
  epidemic, 397
Polycystic disease of kidney, 156
Polycythemia, 163
Polypnea, 237
Polyuria, 122
Pomphi, 550
Pompholyx, 585
Port-wine mark, 598
Precordial prominence, 193
Prickly heat, 584
Progeria, 187
Prosopalgia, 517
Prostration, nervous, 512
Prurigo, 577
Pruritus, 606
Pseudoleukemia, 174
Pseudomuscular hypertrophy, 526
    gait of, 438
Psilosis, 79
Psoriasis, 571
Psychic disturbances, 447
Pulmonary induration, 294
  insufficiency, 218
  stenosis, 218
Pulsation, abnormal centers of, 192
Pulse, 197
  capillary, 202
  Corrigan's, 202
  dicrotic, 202
  increased frequency of, 198
  infrequency of, 198
  tardy, 203
  venous, 202
  water-hammer, 202
Pulses, asymmetric radial, 202
Pulsus alternans, 201
  paradoxus, 201
Pupil, Argyll-Robertson, 446
Pupils, inequality of, 446
Purpura, 176
  hemorrhagica, 177
  Henoch's, 177
  primary, 177
  rheumatica, 177
  Schönlein's, 177
  secondary, 176
Purpuric spots, 543
Pustules, 546
Pyelitis, 149
Pyelonephritis, 149
Pyemia, 374
Pyogenic infections, 374
Pyonephrosis, 150
Pyothorax, 312
Pyromania, 448
Pyuria, 135

RABIES, 401
Rachitic rosary, 241
Rachitis, 419
Radioscopy of chest, 250
Râles, 248
Rashes, date of appearance in acute
  infections, 319
Raynaud's disease, 444, 532
Reaction of degeneration, 443
Reflex, abdominal, 440
  ankle, 439
  arm, 439
  Babinski's, 439
  cremasteric, 440
  gluteal, 440
  headache, 500
  jaw, 439
  Kernig's, 439
  knee, 438
  patellar tendon, 438
  plantar, 440
Reflexes, cutaneous, 439
Reichmann's disease, 51
Relapsing fever, 334
Remittent fever, 343
Renal calculus, 152
Rennet, test for, 23
  zymogen, test for, 23
Resonance, percussion, 244
  vocal, 248
Respiration, 236
  Cheyne-Stokes, 237
  number per minute, 236
  tidal-wave, 237
Respiratory organs, physical examination of, 240
  system, diseases of, 235
Retropharyngeal abscess, 38
  lymphadenitis, 38
Rheumatic fever, 367
Rheumatism, acute articular, 367
  chronic, 531
  muscular, 524
Rheumatoid arthritis, 528
Rhinitis, acute, 251
  atrophic, 253
  chronic, 252

Rhus poisoning, 579
Rickets, 419
Riegel's test-meal, 21
Ringworm, 607
  of body, 609
Robert's nitric magnesium test for albuminuria, 132
Rocky Mountain spotted fever, 406
Rodent ulcer, 604
Romberg's sign, 486
Rose-cold, 274
Roseola, epidemic, 354
Rotch's sign, 207
Rötheln, 354
Round ulcer of duodenum, 54
    of stomach, 54
Round-worm infection, 411
Rubella, 354
Rubeola, 352
Rumination, 25
Rupia, 604

SATURNISM, 537
Scabies, 611
Scalds, 579
Scales, 551
Scarlatina, 347
Scarlet fever, 347
    anginoid, 349
    malignant, 349
Scars on tongue, 19
Sciatica, 496
Sclerema, 594
Scleriasis, 594
Scleroderma, 594
Sclerosis, amyotrophic lateral, 482
  disseminated cerebrospinal, 489
  insular, 489
  lateral, 481
  multiple, 489
  posterior spinal, 485
Scorbutus, 427
Scurvy, 427
Seat-worm, 411
Sebaceous glands, functional diseases of, 556
Seborrhea oleosa, 556
  sicca, 556
  congestiva, 599
Sensation, disturbances of, 440
Sense, muscle, 442
  of space, 441
Senses, special, disturbances of, 446
Septicemia, 374
Septicopyemia, 374
Serositis, multiple, 206
Shaking palsy, 521
Shingles, 565
Ship fever, 332
Sick-headache, 520
Singultus, 26
Skin, blueness of, 540
  cancer of, 604
  chafing of, 560
  dark-brown discoloration of, 540
  diseases of, 540
  emphysema of, 541
  enlargement of superficial veins of, 541
  eruptions of, 542
  glossy, 541
  grayness of, 540
  hardness of, 540
  induration of, 540
  inflammatory diseases of, 560
  pallor of, 540
  trophic affections of, 444
  whiteness of, 540
  yellowness of, 540
Skoda's resonance, 245
Sleeping sickness, 408
Small-pox, 355
Smell, sense of, 235
Sore-throat, acute, 35
    chronic, 37
Space, sense of, 441
Spasm of esophagus, 39
Spasmodic croup, 256
Spasms, 435
Spastic gait, 437
  paralysis of infants, 458
  paraplegia, primary, 481
Spinal cord, diseases of, 450
  leptomeningitis, 474
  meningitis, 474
  muscular atrophy, progressive, 480
  paralysis, acute infantile, 397
  sclerosis, posterior, 485
Spirillum fever, 334
Spleen, chronic enlargements of, 189
Splenic anemia, 189
Splenomegaly, primitive, with anemia, 189
  tropical, 407
Spondylitis deformans, 530
Spondylose rhizomélique, 530
Spongy, bleeding gums, 17
Spotted fever, 336

Sprue, 79
Steatoma, 559
Stellwag's sign, 182
Stenocardia, 226
Stenosis of esophagus, 39
  pulmonary, 218
Steppage gait, 438
Stomach, absorptive, test for, 24
  atony of, 47
  cancer of, 59
  contents, examination of, 21
  dilatation of, 61
    acute 65
  motor insufficiency of, 47
    power of, 24
  neuralgia of, 52
  ulcer of, peptic, 54
    perforating, 54
    round, 54
Stomatitis, 28
  acute, catarrhal, 28
  aphthous, 29
  gangrenous, 30
  gonorrheal, 31
  mercurial, 31
  mycotic, 29
  ulcerative, 29
Stools, 26
  varieties of, 27
Strawberry tongue, 18, 33
Stroke, heat, 533
St. Vitus' dance, 436, 514
Subacidity of gastric contents, 25
Subnormal temperature, 321
Succussion-splash, 250
Sudamen, 555
Sunstroke, 533
Superacidity of gastric contents, 50
Suppurative cholangitis, 97
  encephalitis, 472
Swallowing, difficult, 20
Sweat-glands, diseases of, 554
Swimming in head, 503
Sycosis, 568
  parasitica, 609
Sydenham's chorea, 514
Symmetric gangrene, 532
Syphilis cutanea, 601
Syphiloderm, annular, 603
  bullous, 603
  gummatous, 603
  macular, 602
  papular, 602
  papulosquamous, 603
  pustular, 603
Syphiloderm, tuberculous, 602
Syringomyelia, 491

TABARDILLO, 332
Tabes dorsalis, 485
Tâché cérébrale, 451
Tachycardia, 198
Tænia echinococcus, 410
  saginata, 409
  solium, 409
Tallqvist's hemoglobinometer, 157
Tapeworm, broad, 409
  dwarf, 410
  fat, 409
  infection, 409
  pork-measle, 409
Teeth, 17
  Hutchinson's, 17
Temperature, subnormal, 321
Test-breakfast of Ewald and Boas, 21
Test-meal, Riegel's, 21
Tetanus, 395
Tetany, 187
Tetter, 573
Thermic fever, 533
Thermo-anesthesia, 441
Thomsen's disease, 527
Thrombosis, cerebral, 466
  of mesenteric vessel, 121
Thrush, 29
Tic, 436
Tic douloureux, 517
Tinea circinata, 609
  favosa, 611
  sycosis, 569, 609
  tonsurans, 608
  versicolor, 610
Tinnitus aurium, 447
Titubation, 438
Tongue, 17
  black, 18
  discoloration of, 18
  dry, brown, 18
  enlargement of, 18
  fissured, 18, 19
  fur on, 17, 18
  red, beefy, 18
  scars on, 19
  strawberry, 18
  tremor of, 19
Tonsillitis, acute, 31
  follicular, 32
  phlegmonous, 32
Tonsils, hypertrophy of, 34

Tophi, 216
Tormina, 70
Torticollis, 435, 524
Toxemic headache, 500
Traube's semilunar space, 244
Tremor of tongue, 19
Tremors, 437
Trichina spiralis, 413
Trichiniasis, 413
Tricuspid insufficiency, 217
  stenosis, 217
Trifacial neuralgia, 517
Trousseau's sign, 188
Trypanosomiasis, 408
Tubercles, 549
Tuberculosis, acute general, 383
    pneumonic, 300
  chronic fibroid, 302
    ulcerative, 300
  miliary, 383
  of kidney, 155
  pulmonary, 297
Tumors of brain, 467
  of kidney, malignant, 156
Tylosis, 591
Typhlitis, 79
Typhoid fever, 322
    varieties of, 326
    Widal reaction in, 326
  pneumonia, 286
Typhus abdominalis, 322
  fever, 332
Tyrosinuria, 124

ULCER, peptic, 44
  rodent, 604
Ulceration from perverted nutrition, 443
Ulcers, 552
Uncinaria americana, 412
  duodenalis, 412
Uncinariasis, 412
Urea excretion, 122
Uremia, 139
  latent, 140
Uric acid diathesis, 416
Urine, 122
  chlorids in, 127
Urobilinuria, 128
Urticaria, 562
  giant, 563
  hæmorrhagica, 563
  papulosa, 563
  tuberosa, 563

VACCINATION, 360
Vaccinia, 360
Valleix's points douloureux, 516
Valvulitis, 209
Vaquez' disease, 178
Varicella, 361
Variola, 355
Venereal wart, 593
Verruca, 592
  acuminata, 593
  digitata, 593
  filiformis, 593
  plana, 593
  vulgaris, 593
Vertigo, 503
Vesicles, 544
Vincent's angina, 36
Vitiligo, 586
Vitiligoidea, 598
Voice, loss of, 236
Vomit, 20
  varieties of, 20, 21
Vomiting, 20
Von Graefe's sign, 182
Von Recklinghausen's disease, 597

WART, 592
  venereal, 593
Water on brain, 454
Waxy kidney, 148
  liver, 113
Weil's disease, 115
Weir Mitchell's rest-cure, 513
Wen, 559
Westphal's sign, 486
Wet brain, 535
Wheals, 550
Whooping-cough, 384
Widal reaction in typhoid fever, 326
Winter cough, 265
Worm infection, 409
Wrist-drop, 538
Writer's cramp, 523
Wry-neck, 435, 524

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  fever, 380

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Zuckergussleber, 108

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